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SPONTANEOUS RUPTURE OF THE STOMACH DURING LABOR*

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SEARCH of the literature and conversation with surgeons and obstetricians of wide experience have led me to believe that the case which I shall report is quite unique, namely, the spontaneous rupture of the stomach during labor with lesions of the liver suggestive of eclampsia.

This patient, a twenty-nine year old primipara, a physician's wife, came under observation at her fourteenth week of pregnancy. Scarlet fever at five years, a questionable attack of typhoid fever and an appendectomy were the only items of interest in her past history. Since her weight, 139 pounds, was about 10 pounds above her ideal, she kept herself carefully on a reduced general diet, and at term weighed only 141 pounds, vitamin deficiency being guarded against. General physical and pelvic examinations showed no abnormalities, nor did the urine and blood pressure deviate from the normal on seven prenatal visits. The last examination, two weeks before delivery, showed no albumen or casts, and the blood pressure was 132/75. Her general health was excellent during pregnancy except for considerable "heart burn" and some vomiting during the last month. Neither her husband nor I felt that this was marked enough to be of significance, though it caused her considerable discomfort.

Because of this discomfort, on February 7 three days before her calculated term, she was given one ounce of castor oil and ten grains of quinine bisulphate. She did not fall into labor and, unknown to me, she repeated the dose on the following morning, 46 hours before delivery, entering the hospital that evening in labor. Regular pains at five minute intervals were present at 8:00 P.M., the presentation being L. O. P. head at the spines, cervix one finger dilated and thick.

Pains increased in frequency, regularity and strength, and at 1:00 A.M. on February 9, 30 hours before delivery, she was given six grains of pentobarbital and at 1:30 A.M. six drachms of paraldehyde in one and one-half ounces of sweet oil, by rectum. She obtained much needed rest and sleep but labor was interrupted and practically ceased until 10:00 A.M.

Catheterization produced 425 cc. of urine at 5:30 A.M. and shortly thereafter she vomited twice small amounts of yellow and green fluid. Toward noon, 17 hours before delivery, labor pains of fair quality began again, reaching five minute intervals by 6:00 P.M. She voided at 10:00 A.M., 2:00 P.M. and 6:30 P.M. She vomited her light supper at 5:30 P.M.

During the evening of February 9, however, labor set in more vigorously. At 10:00 P.M., seven hours before delivery, she was given six grains of pentobarbital by mouth, and a Buist roll was applied on the side of the child's back, over the left Poupart's ligament. The cervix at this time was two fingers dilated. Very little analgesia was produced by the pentobarbital, and at 12:50 A.M. I gave her three grains more by rectum. Beginning at 10:00 P.M., she was given gas oxygen with her pains which were severe and very painful.

Because of her vomiting and the prolongation of labor without adequate food or fluids, I gave her 700 cc. of five per cent glucose in saline intravenously, beginning at 1:00 A.M. There was something indefinable about her appearance which caused me anxiety, though there was nothing to be noted which differed from many other patients in whom labor was prolonged.

At 2:30 A.M. on February 10, vaginal examination showed three fingers' dilation. The membranes which had descended into the vagina were artificially ruptured, but the head could not, with the fingers, be rotated to L. O. A. The Buist roll was removed and a Beck labor corset was applied, with marked improvement in uterine contractions. (The Beck corset fitted over the entire pregnant uterus snugly, but did not compress the upper abdomen or interfere with ease of respiration.)

The end of labor seemed to be a matter of only a few more hours, when at 3:15 A.M., two hours before delivery, she suddenly had a severe chill lasting twenty minutes and vomited 100 cc. of brown fluid. Her pulse rose to 160, she seemed very exhausted and showed a cyanotic pallor which was very alarming.

After allowing a partial recovery from this condition, I was able to deliver her, under ether anesthesia, with single application of forceps, L. O. T., cervix incision (3 cm. in the midline), and with perineotomy. The child was easily delivered in good condition, weighed eight pounds seven ounces, and has developed normally to date.

The cervix showed no tears, and the incision was repaired easily. The placenta separated naturally and was expressed from the vagina. The fundus contracted well, and one cc. of pituitrin and 1/6 of morphine were given. She was then placed in shock position, and I considered her out of danger though still in need of careful treatment following her severe labor.

I was called back to the delivery room from which the patient had not been moved, about one hour after delivery. The pulse had disappeared and she was in deep shock, 1/6 grain morphine having been repeated. A few clots had been expressed from the vagina but the fundus was well contracted. Pituitrin and gynergen, one ampoule of each, were given though the blood loss was very moderate. Preparations were made for a transfusion. One thousand cc. of five per cent glucose which had been given by rectum following delivery had been retained and another 1/6 grain of morphine was given because she was very restless. This made 1/2 grain of morphine in the space of two hours.

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Consultation was had with Dr. T. W. Worthen and Dr. Robert Starr at 9:00 A.M., lungs and heart being ruled out as a cause of the collapse. Further morphia, fluids and transfusion were suggested.

During the next hour 1500 cc. of saline and glucose five per cent were given with 15 drops of adrenalin 1-1000, with marked improvement. At 1:30 P.M., blood pressure was 104 systolic, pulse and general appearance were much improved and the patient was conscious. At 3:00 P.M., ten hours after labor, she collapsed again, and was given intravenous saline and glucose with 400 cc. of blood. Following this, she looked in excellent condition, took an interest in her baby, and sodium luminal, grains 1/2 s. c. was given as a substitute for morphine.

At about 7:00 P.M. she collapsed again. Drs. Townsend and MacClellan saw her in consultation and arrangements were made for a second transfusion and intravenous fluid administration. The urine output at 4:30 P.M. by catheter totaled 200 cc. and showed a large amount of albumen with hyalin and cell casts, alkaline reaction, with a few white and red blood cells. During the afternoon she began to vomit and showed considerable soft, doughy distention in the upper abdomen. A Levine tube was passed and a large amount of coffee-ground and blood-tinged fluid was obtained. She also began passing blood-tinged fluid by rectum.

She died shortly after midnight, 19 hours after delivery, having lapsed into unconsciousness about one hour before death. Râles throughout the chest and embarrassed respiration were observed.

Before turning to the autopsy findings, let me summarize the salient features.

A twenty-nine year old primipara, moderate epigastric pain and vomiting of one month's duration, medical induction of labor at term, quinine bisulphate, grains 10, and castor oil, one ounce, on two successive mornings, labor lasting 52 hours. Analgesia by pentobarbital, by mouth 2 x 6 grains and by rectum 3 grains, and paraldehyde 6 drachms. Had gas-oxygen with pains for five hours, ether to surgical anaesthesia; albumen 3 plus, and cell casts in catheterized specimen during labor. Prolonged L. O. P. with slow progress and collapse two hours before full dilatation, forceps delivery, further and repeated collapse of circulatory type, transfusions, distention of upper abdomen and death. Use of Buist roll and Beck corset during labor.

AUTOPSY (Dr. Wilmar M. Allen)

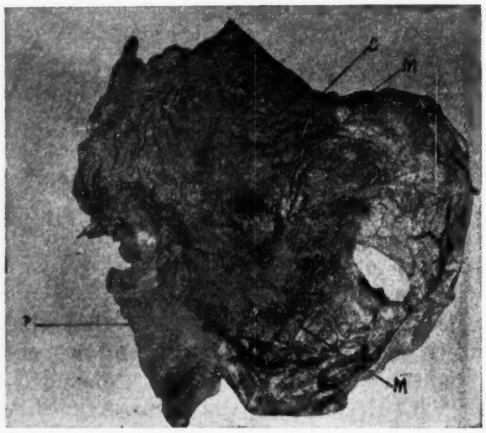
Summary of gross—Young adult white woman, ten hours postpartum. Pulmonary edema. Acute generalized peritonitis with butyric acid odor. Superficial ulceration of esophagus. Large perforation of stomach with retraction of mucosa. Acute nephrosis, hydroureter, and hydronephrosis, right, moderate. Puerperal genitalia.

Impression from gross: Acute rupture of stomach with generalized peritonitis.

Anatomical diagnosis:

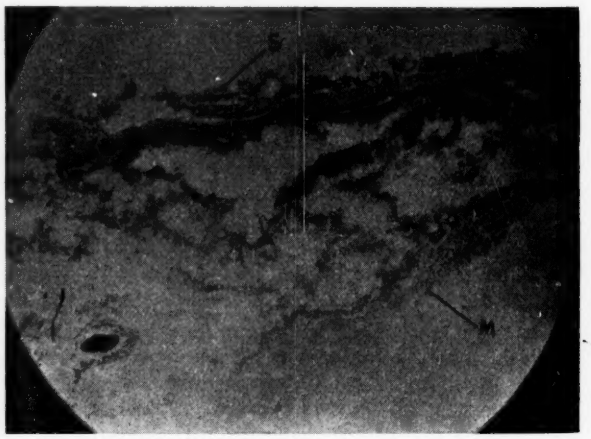
1. Eclampsia gravidarum (?) with acute perforation of stomach and acute generalized peritonitis (B. coli and unidentified gram positive anaerobe).
2. Acute nephrosis.
   Hydroureter and hydronephrosis, right.
   Pulmonary edema.

General description: There is a moderate brownish frothy discharge from nose and mouth. The abdomen is considerably distended and tympanitic. The peritoneal cavity contains about 2 litres of moderately turbid, definitely blood-tinged fluid with a definite butyric acid odor. There is general hyperemia and a very thin layer of fibrin. The peritonitis is absolutely generalized. The esophagus appears normal, except for rather marked hyperemia and superficial ulceration near the cardia. The stomach is not distended. It is examined very gently and carefully in situ. It shows a large ragged perforation about three cm. in diameter on the greater curvature at about the mid portion. It is just posterior, but a portion of the perforation involves the omentum, and gastric contents are dissecting into the omentum. The lesser peritoneal cavity contains a moderate amount of coffee-ground material which is not present throughout the peritoneal cavity.



1. Shows perforation in the stomach wall on the greater curvature nearer the cardiac than pyloric end of the stomach. The wide area of retracted mucosa can be seen, marked "M". "P" Pylorus—"C" Cardia.

On opening the stomach the perforation is seen to have a ragged frayed margin. It is roughly circular. The margins are brownish and show some digestive action, but the perforation appears to be not more than 24 hours old and no evidence of an old lesion can be found.



2. Low power view of the ulcer edge showing no inflammatory reaction of a recent or scar tissue reaction of an old ulcerative process. "M" Mucosa—"S" Serosa.

In addition to the perforation there is apparently an extraordinary retraction of the mucous membrane. This forms a roughly diamond-shaped area extending from the cardia almost to the pylorus and about 10 cm. in width in the center. The mucosa at the margin of this area is closely and smoothly applied to the underlying coat. It shows no hemorrhage or ulceration. This area apparently is covered only by serosa, which is quite clean and fresh looking. No necrosis or inflammatory reaction. It has the appearance of a quite fresh tearing of the stomach wall, except that the margin of the mucosa ap-

pears smoothly organized to the underlying structures.

The remainder of the stomach appears normal, as does the duodenum. The small intestine is considerably distended and hyperemic, but nowhere obstructed or gangrenous. The liver appears entirely normal. There are no eclamptic lesions. The kidneys are moderately enlarged, weighing together 440 Gm. The capsule strips readily, leaving a smooth surface. The cortex averages 14 mm. The architecture appears intact, but there is a general, fairly marked, cloudy swelling. The cortex is slightly yellowish white in color. The pyramids are considerably congested and afford a sharp contrast. The renal vessels appear normal. The pelvis of the right kidney is considerably dilated, and there is apparently no diminution of parenchyma. There is no evidence of inflammation. There is a large aberrant vessel to the lower pole, but this does not produce any apparent obstruction. The right ureter is likewise considerably dilated. From the brim of the pelvis to the bladder the calibre is normal. There is no abnormality in the ureteral wall to account for the obstruction. It is dilated and shows no evidence of inflammation. The genitalia are typically puerperal. The uterus is firmly contracted and appears entirely normal except for three tiny fibroids about 5 mm. in diameter. The cavity contains a small amount of firmly adherent blood clot. The placental attachment was posterior. There is no evidence of infection. Tubes and ovaries are normal.

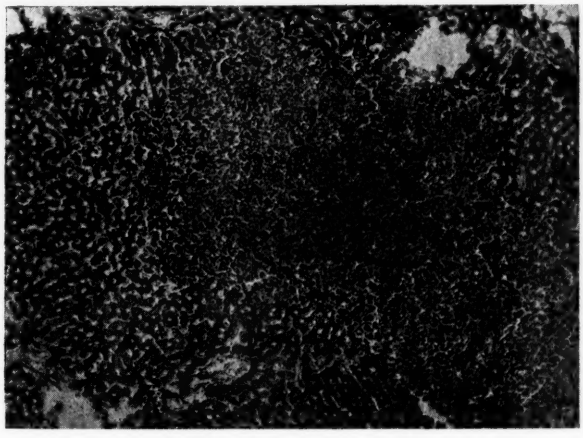
There is slight ecchymosis of the conjunctivae and the retroperitoneal tissues exhibit a moderate edema. In the serosa of the intestines there are small gas bubbles but these are not present elsewhere and are apparently associated with the peritonitis, which, as noted above, has a strong butyric acid odor.

Microscopic Findings:

Heart: General structure and cytology well preserved. No inflammatory reaction, edema or obvious degeneration.

Lungs: Moderate edema.

Spleen: Shows slight congestion and increase in pulp cells.

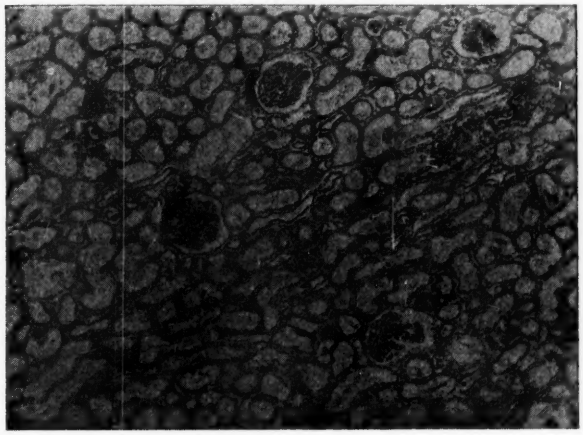


3. Shows periportal liver necrosis. In no section could thrombosis be demonstrated.

Stomach: Sections around the perforation show the margins to be absolutely clean-cut. There is no inflammatory reaction and no evidence of an old process. It appears to be a quite fresh mechanical rupture of the stomach wall. In places the submucosa is edematous. Outside of the area of perforation, the gastric wall appears essentially normal.

Pancreas: Normal.

Liver: Shows rather striking changes which, however, are difficult to interpret. There is a rather widespread zonal degenerative process around the efferent vein. In some areas the cells are preserved, but have lost their trabecular arrangement and seem to be sitting isolated in groups. In other areas these cells show quite marked cloudy swelling and degenerative changes. There are in addition rather numerous definite focal necroses, some of which are periportal. In none of the organs are the red blood cells preserved, so that hyaline thrombi cannot be made out. These liver lesions, however, are extremely suggestive of an eclamptic process.



4. Shows moderate cloudy swelling of the kidney, dilatation of the tubules, with occasional casts and well-preserved glomeruli.

Kidneys: There is fairly marked dilatation of the tubules. A number contain serum and casts. The epithelium is thin and shows fairly marked, cloudy swelling. There are no hemorrhages, glomeruli appear normal. No vascular changes. No inflammatory reaction.

Speculation as to the cause of the rupture of the stomach leads at once to a consideration of the mechanical factors involved in labor by the strain of vomiting, by the labor corset, or both, with or without the hypothetical weakness of the stomach wall.

Certainly the time of acute rupture can be placed at the time of the first collapse. This occurred three-quarters of an hour after applying the labor corset. The patient was not being encouraged to bear down with her pains at this time, though she was being given gas. One can easily imagine, however, that she did try to bear down and help labor along. It can thus be assumed that either by vomiting or by straining, with a laboring full term uterus supported by a labor corset, the intraäbdominal pressure was greatly increased. Just how this would tend to burst the stomach, however, it is difficult to see, for the pressure within the stomach should be as great as on its peritoneal surface.

As to the drugs, castor oil and quinine can, I believe, be dismissed, and also one can recall no liver lesion such as this due either to barbiturates or to paraldehyde.

It is to be regretted that no examination was made of the brain, for the stomach findings at autopsy, as well as the history of sudden onset, remind one of some of the cases presented by Dr. Harvey Cushing. In his article "Peptic Ulcer and the Interbrain", the neurogenic origin of peptic ulcer was discussed at length, and what appeared to be incontrovertible evidence was presented, showing relationship between the stomach lesion and the lesions in the interbrain. These lesions in the interbrain were of such a nature as to interrupt the continuity of the parasympathetic nerve fibres, allowing a loss of balance between the parasympathetic and vagus influence on the stomach.

In retrospect it is impossible to recall any symptoms suggestive of a brain lesion, though vomiting during the last month of pregnancy may well have been an expression of overactivity of the vagus. It is interesting to note that our patient had large doses of barbiturates which, according to Cushing, inhibit the vagotonic hyperactivity of the stomach as demonstrated in experimental work.

In the absence of a brain examination we can merely present the case as a medical curiosity and suggest that the report of similar cases may some day furnish an adequate explanation.

REFERENCES

Cushing, Harvey: Peptic ulcers and interbrain. Surg. Gynec. Obst. 55:1 (July) 1932.

DISCUSSION

DR. CHARLES L. SCUDDER, Boston, Mass.: Dr. Miller did not say whether there was evidence of ulcer there.

DR. MILLER: The microscopic slide shows no leukocyte infiltration, no scar tissue, and in the gross absolutely no sign of a previous ulcerated lesion. The extraordinary thing about it is the remarkable retraction of the mucosa over an area as large as 10 by 20 centimeters, almost from the cardia, two-thirds of the way to the pylorus. It looks like an explosive and a digestive lesion.

The autopsy was ten hours postmortem. We had this definite catastrophe occurring before delivery.

DR. STEPHEN A. MAHONEY, Holyoke, Mass.: Was the stomach full or empty?

DR. MILLER: Some vomiting had occurred in the last hours before the delivery and she vomited during labor. The stomach contained some coffee-ground material only, no food.

THE GEOGRAPHIC DISTRIBUTION OF HOMICIDE IN THE UNITED STATES

Homicide, in its wide prevalence throughout the United States, presents one of the unsolved social problems of the country. Why should the homicide rate in our white population be three times as high among us as across the border among our neighbors in Canada, and ten or eleven times as high as in England? Several circumstances, no doubt, contribute to this effect, but for the time being we must be satisfied with accepting the fact which confronts us.

Of all the states, Nevada had the highest homicide rate among white persons, namely, 16.5 per hundred thousand, during the years 1929 to 1931; the average rate for the country as a whole being 5.5. For colored people, the unenviable distinction of heading the list falls to Missouri, with a rate of 72.2 per hundred thousand, the average for persons of this racial group being 38.4. The lowest homicide rates, on the other hand, were recorded in Maine and Vermont for white persons, each with a rate of 1.3 per hundred thousand, and in New Hampshire and Vermont for colored persons, where no deaths from this cause were recorded in any of the three years under consideration.

Above-average homicide rates are concentrated in certain areas of the country. For the white population, the region of highest homicide rates forms two patches, one in the Southeast, and one mainly in the Mountain States, with the State of Oklahoma forming a connecting link.

In the southeastern area, particularly high rates among white persons (over 10 per 100,000) are recorded in the States of Florida, Kentucky and Mississippi. Rates between 8 and 10 per 100,000 are recorded in West Virginia, Tennessee, South Carolina, Georgia, Alabama, Louisiana and Arkansas.

In the Mountain State region, very high rates are found in Nevada, Arizona and New Mexico. Montana and Wyoming have rates over eight per hundred thousand.

The New England States are distinguished by very low homicide rates. The adjacent Middle Atlantic States also, New York, New Jersey and Pennsylvania, fall below the average for white persons.

It should, of course, be realized that homicide is mainly an urban problem. Rural areas show uniformly lower rates.—*October Bulletin, Metropolitan Life Insurance Company.*

DR. WYNNE'S OPPOSITION TO EYEBROW OR EYELASH DYES

The action of Dr. Shirley W. Wynne, Health Commissioner of New York City, in prohibiting the manufacture, sale or use of eyebrow or eyelash dyes which are known to contain harmful ingredients, was commended today by Lewis H. Carris, Managing Director of the National Society for the Prevention of Blindness.

"If only one woman in New York City should be spared the tragedy of blindness through Dr. Wynne's prompt order as soon as this menace to eyesight was disclosed, his action would be entirely justified," said Mr. Carris. "In a city as large as New York —with its thousands of beauty parlors patronized by perhaps a million or more women—the chances of injury to eyesight, if there is no law against the use of ingredients known to be harmful, are quite obvious."

The Sight-Saving Review, quarterly journal of the National Society for the Prevention of Blindness, in its December issue, will comment editorially on the danger to women's eyes from the use of irritating dyes around the delicate tissues of the eye, Mr. Carris said.—*Bulletin of the National Society for the Prevention of Blindness.*

THE USE OF SODIUM DEHYDROCHOLATE AS A CLINICAL TEST OF THE VELOCITY OF BLOOD FLOW*

BY SAMUEL L. GARGILL, M.D.†

INTRODUCTION

IN a series of studies on the velocity of blood flow Blumgart and his associates[1] described a safe and accurate method for measuring the velocity of blood flow and stressed the clinical significance of such measurements in health and disease. The results of velocity studies offer definite diagnostic and prognostic aid in many pathological conditions, especially in diseases associated with dyspnea and edema. Following the studies of Blumgart, a widespread interest in the clinical aspects of the velocity of blood flow has developed, and several simple methods of measurement at the bedside have been described. Before starting the present investigation, a critical survey of these simple methods was made and the method recently described by Winternitz and his associates[2] was chosen as the most suitable for clinical use. These investigators used an aqueous solution of sodium dehydrocholate, marketed as "Decholin," as the test material for intravenous injection.

In the present study, a slight modification of the decholin method of Winternitz is described, and the accuracy and general dependability of this decholin method as applied in the normal individual and in patients with various pathological circulatory conditions have been investigated.

METHOD EMPLOYED

Winternitz[2] and his associates injected five cc. of a twenty per cent solution of decholin into an arm vein and measured the time that elapsed between the end of the injection and the appearance of a bitter taste on the tongue. As this taste disappeared within a few seconds, the test could be readily repeated and checks of the measurement obtained. Winternitz found that in normal individuals an intense bitter taste appeared in eight to fourteen seconds after the end of the injection. In various pathological circulatory states striking deviations from the normal were found, agreeing in general with the findings by the more accurate radioactive method[1]. Winternitz observed no untoward effects from the injection of five cc. of decholin. The injection of larger amounts produced in a few individuals transient epigastric distress and nausea.

Because the intensity of the pharmacodynamic effect of a substance depends on the concentration in which it comes in contact with the tissues, an attempt was made to use smaller volumes of more concentrated solutions of decholin. The injection of a smaller volume than that used by Winternitz also seemed advantageous since the time necessary for the injection would be less prolonged. The use of decholin in concentrations of from thirty to fifty per cent, however, proved unsuccessful, since these higher concentrations of decholin were unstable and produced undesirable clinical effects. It was found, however, that three cc. of a twenty per cent solution of decholin produced the characteristic bitter taste in all subjects. The time necessary for injection of this smaller amount through an 18 gauge needle was usually only two to three seconds. The injection time could be shortened to one second or less by using a 16 gauge needle. In Winternitz's studies the circulation time from the end of injection was recorded, no injection time being given. In the present study the circulation time is measured from the beginning of the injection and the injection time is recorded separately.

The following method was, therefore, adhered to throughout the present investigation. All tests were performed with the subjects in the recumbent position and under strict metabolic conditions. The contents of a ten cc. ampule of twenty per cent solution of decholin were aspirated in a ten cc. sterilized Luer syringe. A vein in the antecubital space of the arm was entered and three cc. of decholin were injected as rapidly as possible. The time taken for injection as well as the time of onset of the bitter taste were registered by means of a stop watch. The time elapsed from the beginning of injection to the onset of the bitter taste was recorded as the "arm to tongue circulation time." After an interval of one or two minutes, with the needle still in place, another three cc. of decholin were injected and the circulation time was again measured. The last four cc. of decholin in the syringe were used to obtain a third measurement. These three measurements usually checked within one second or a fraction thereof.

RESULTS

The arm to tongue circulation time in normal individuals—The arm to tongue circulation time was measured with decholin in 50 normal subjects. Table I presents the results of these measurements according to the age incidence of the subjects. It is apparent that age has no influence on the velocity of blood flow. The arm to tongue circulation time measured from the beginning of injection varied from fifteen to twenty seconds; the average circulation time was eighteen seconds, and the average injection time

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was three seconds. The average actual circulation time, therefore, was fifteen seconds. This compares favorably with the arm to arm circulation time measurements by the radioactive method which, in 53 normal individuals, varied between fourteen and twenty-four seconds, the average circulation time being eighteen seconds[5]. The slightly shorter average circulation time obtained by the decholin method can be explained by the fact that the arm to tongue pathway is somewhat shorter than the arm to arm pathway.

The jugular to tongue (pulmonary) circulation time in normal individuals—The velocity of blood flow through the lungs was found by Blumgart and Weiss[3] to vary less in a given individual than the velocity of blood flow through a peripheral pathway. Studies of the pulmonary circulation time were accordingly made in twelve normal individuals by injecting the decholin into the external jugular vein following the method of Robb and Weiss[4]. The measurement thus obtained was recorded as the "jugular to tongue circulation time."

TABLE I

DECHOLIN ARM TO TONGUE CIRCULATION TIME IN FIFTY NORMAL INDIVIDUALS

Age	Number of Subjects	Heart Rate	Decholin Injected	Circulation Time			Injection Time	Actual Circulation Time
				Minimal	Maximal	Average		
years		average	cc.	seconds	seconds	seconds	seconds	seconds
20-29	8	76	3	16	19	18	3.0	15
30-39	10	80	3	15	18	17	3.0	14
40-49	13	78	3	15	20	17	2.8	14.2
50-59	9	84	3	16	19	18	3.0	15
60-69	10	76	3	17	20	18	2.6	15.4

TABLE II

DECHOLIN PULMONARY CIRCULATION TIME IN TWELVE NORMAL INDIVIDUALS

Case	Age	Heart Rate	Decholin Injected	Jugular to Tongue Circulation Time	Injection Time	Actual Circulation Time
	years		cc.	seconds	seconds	seconds
1	26	72	3	12.5	1.5	11
2	30	80	3	13.0	1.0	12
3	32	84	3	13.5	2.0	11.5
4	20	80	3	12.0	2.0	10
5	44	76	4	14.0	2.0	12
6	52	68	3	14.5	1.8	12.7
7	48	72	3	12.6	2.0	10.6
8	60	72	4	14.0	1.5	12.5
9	58	76	3	12.0	1.0	11.0
10	54	76	3	13.2	2.0	11.2
11	31	68	3	15.0	2.0	13
12	36	72	3	12.6	1.5	11.1
Average	40.9	74.7	3.2	13.2	1.4	11.8

The results of these measurements are given in table II. The jugular to tongue circulation time varied from twelve to fifteen seconds; the average circulation time was thirteen and two tenths seconds, and the average injection time was one and four tenths seconds. The average actual jugular to tongue circulation time, therefore, was eleven and eight tenths seconds. It is of interest that the pulmonary circulation time measured by the radioactive method in 62 normal subjects varied from five to seventeen seconds, with an average of ten and eight tenths seconds. The injections into the external jugular vein proved as simple as the injections into the antecubital vein of the arm. In two apprehensive individuals it was considered advisable to infiltrate the skin over the jugular vein with novocain preliminary to the injection. No untoward reactions were observed in any of the subjects and there were no significant changes in the heart rate or the blood pressure level.

Comparison of circulation times as determined by the cyanide and decholin methods in normal individuals—Because the reaction to decholin is subjective in nature, it was thought advisable, in order to test further the accuracy of the method, to compare it with the cyanide method recently described by Robb and Weiss[4]. This method makes use of an objective reaction produced by the intravenous injection of two per cent sodium cyanide and has an end point signalized by a sudden increase in the depth and rate of respiration. Comparative circulation time measurements with this method and with the decholin method were performed in fifteen normal subjects. With the subject under strict metabolic conditions four tenths cc. of a two per cent solution of sodium cyanide were injected into the antecubital vein of one arm, and the time elapsing between the injection and the appearance of the sudden respiratory gasp was registered by means of a stop watch. After an interval of five minutes three cc. of a twenty per cent solution of decholin were injected into the vein of the other arm, and the injection time, as well as the circulation time, was similarly recorded. The results of this comparative study are shown in table III. The circulation time with the cyanide method varied from twelve and five tenths to eighteen seconds with an average of fifteen and six tenths seconds. The circulation time with the decholin method varied from fifteen to twenty seconds with an average of seventeen and five tenths seconds. The average

injection time of the decholin was one and six tenths seconds. If this is subtracted from the average "arm to tongue circulation time", it leaves an actual circulation time of fifteen and nine tenths seconds which compares very well with the average cyanide circulation time of fifteen and six tenths seconds.

Decholin circulation time in various pathological states—The "arm to tongue circulation time" was measured with decholin in five groups of patients showing clinical evidence of the following disorders: (a) hyperthyroidism, (b) secondary anemia, (c) arterial hypertension, (d) pulmonary emphysema, and (e) congestive failure. The results are summarized in table IV, and will be discussed under separate headings in accordance with the diagnosis of each group.

TABLE III

COMPARISON OF CIRCULATION TIMES AS DETERMINED BY THE CYANIDE AND DECHOLIN METHODS IN FIFTEEN NORMAL INDIVIDUALS

Case	Sodium Cyanide		Decholin			
	Amount Injected	Circulation Time Arm to Carotid Sinus	Amount Injected	Circulation Time Arm to Tongue	Injection Time	Actual Circulation Time
	cc.	seconds	cc.	seconds	seconds	seconds
1	0.4	14.0	3	17.0	1.5	15.5
2	0.4	15.5	3	16.5	1.0	15.5
3	0.4	14.5	3	16.0	2.0	14.0
4	0.4	12.5	3	15.0	1.6	13.4
5	0.4	17.0	3	18.0	1.2	16.8
6	0.4	14.0	3	16.5	2.0	14.5
7	0.4	17.8	3	19.0	1.5	17.5
8	0.4	16.5	3	18.0	1.2	16.8
9	0.4	13.8	3	16.0	2.0	14.0
10	0.4	18.0	3	19.5	2.0	17.5
11	0.4	16.0	3	18.0	1.5	16.5
12	0.4	14.0	3	17.0	2.0	15.0
13	0.4	18.0	3	20.0	1.5	18.5
14	0.4	15.6	3	17.0	1.2	15.8
15	0.4	16.8	3	19.0	1.8	17.2
Average	0.4	15.6	3	17.5	1.6	15.9

(a) *Decholin circulation time in hyperthyroidism*—The "arm to tongue circulation time" was studied in twelve patients with the classical signs of Graves' disease. The average heart rate was 107, and the average basal metabolic rate was thirty-one per cent above the normal. The circulation time varied from eight to fourteen seconds; the average circulation time was eleven and five tenths seconds, and the average injection time was three seconds, making an actual circulation time of eight and five tenths seconds. In a similar group of patients, Blumgart and his associates[6] obtained an average arm to arm circulation time of ten and eight tenths seconds.

(b) *Decholin circulation time in secondary anemia*—The "arm to tongue circulation time" was measured in ten patients with anemia secondary to recent loss of blood. The average heart rate was 90, the average hemoglobin was forty-six per cent by the Sahli Method, and the average erythrocyte count was three millions per cu. mm. of blood. The velocity of blood flow in this group varied from eight to fourteen seconds, with an average of eleven seconds and an actual circulation time of eight seconds. In a group of patients with secondary anemia, Blumgart and his associates[7] found, with the radioactive method, that the velocity of blood flow was likewise increased.

(c) *The decholin circulation time in arterial hypertension*—The velocity of blood flow was measured with decholin in twelve patients with arterial hypertension who showed no clinical evidence of congestive failure. The average pulse rate was 82, and the average blood pressure was 226 mm. of mercury systolic, and 122 mm. of mercury diastolic. The "arm to tongue circulation time" in these patients varied from seventeen to nineteen seconds, with an average of eighteen seconds, and an actual circulation time of fifteen seconds. The finding of a normal circulation time in these patients is in agreement with the observations of Blumgart and Weiss[8].

(d) *The decholin circulation time in pulmonary emphysema*—The "arm to tongue circulation time" was measured by means of decholin in nine patients with pulmonary emphysema. Although these patients were ambulatory, they all complained of marked dyspnea, and showed marked cyanosis, a positive Hoover's sign, and marked reduction in the vital capacity of the lungs. As summarized in table IV, the circulation times were well within the limits of normal, the average actual circulation time being thirteen seconds. The physical disability in these patients was due, therefore, to the failure of the respiratory function of the lungs

TABLE IV

DECHOLIN CIRCULATION TIME IN VARIOUS PATHOLOGICAL STATES

Number of Patients	Diagnosis	Heart Rate average	Circulation Time Minimal seconds	Circulation Time Maximal seconds	Time Average seconds	Injection Time Average seconds	Actual Circulation Time seconds	Remarks
12	Hyperthyroidism	107	8	14	11.5	3	8.5	Average B. M. R. = +31
10	Secondary Anemia	90	8	14	11.0	3	8.0	Average R. B. C. = 3,000,000 Average Hgb. = 46%
12	Arterial Hypertension	82	17	19	18.0	3	15.0	Average B. P. = 226/122
9	Pulmonary Emphysema	86	14	20	16.0	3	13.0	Average V. C. = 1,570 cc.
10	Congestive Failure	88	25	52	36.0	3	33.0	Two patients had rheumatic heart disease. Eight patients had arteriosclerotic heart disease.

and not to circulatory failure. The finding of a normal velocity of blood flow in patients with pulmonary emphysema and its clinical significance was first pointed out by Weiss and Blumgart[9].

(e) *The decholin circulation time in patients with clinical evidence of congestive failure*—The "arm to tongue circulation time" was measured with decholin in ten patients with symptoms and signs of gross congestive failure. Two of the patients had rheumatic heart disease, while the others had arteriosclerotic heart disease. The circulation time varied from twenty-five to fifty-two seconds; the average actual time was thirty-three seconds (table IV). These observations are in accord with those made in similar patients by Blumgart and Weiss[10] who obtained an average arm to arm circulation time of thirty-eight seconds.

DISCUSSION

In 1927, Blumgart[1] formulated certain requirements for any substance that is to be used for measuring the velocity of blood flow. Briefly, these requirements are 1. The substance must not be toxic in the amounts utilized. 2. The substance should not influence the velocity of blood flow. 3. The substance should disappear from the body with sufficient rapidity to allow repeat measurements.

The present study demonstrates that decholin fulfills these requirements in the following way: 1. In the amounts utilized, decholin produced no untoward effects in over 150 patients. The optimum dose proved to be three cc. This amount of decholin could be repeatedly injected without producing any toxic effects. In a few subjects with marked arteriosclerosis or congestive failure, larger amounts of decholin were required to produce the characteristic bitter taste. These subjects experienced fulness in the epigastrium and nausea of a few minutes' duration. 2. Decholin did not influence the velocity of blood flow. In none of the subjects studied was there any significant alteration in the pulse rate or the blood pressure level. 3. The bitter taste produced by decholin disappeared in fifteen to twenty-five seconds in normal individuals and in about thirty seconds in patients with congestive failure. Experience has shown that in order to obtain the best checks, it is advisable to wait at least one minute in normal subjects, and two or three minutes in patients with congestive failure, before repeating the measurement.

The decholin method of measuring the velocity of blood flow offers certain advantages over the other methods that are available at present. The test can be used at the bedside and requires no complicated apparatus, and no special skill on the part of the physician. The reaction produced by decholin lasts only a few seconds and, therefore, the test can be repeated within a min-

ute or two. Furthermore, decholin is easily available in sterile stable solution.

The three cc. volume of decholin necessary for the measurement can, according to our most recent experience, be injected in less than two seconds. Therefore, the injection time cannot affect appreciably the accuracy of this method. Because of the subjective nature of the response on which the decholin test depends, it was thought that the test might not be so accurate as other tests with objective end points. Comparison of this test, however, with the cyanide method, which depends on an objective response, showed that the results were practically identical in normal individuals. A failure of response has occurred in only three out of more than 150 individuals studied.

CONCLUSIONS

1. A study of the use of decholin injection as a clinical test of the velocity of blood flow is presented.
2. A technic is described which differs in certain respects from the original technic used by Winternitz and his associates.
3. In 50 normal individuals the average "arm to tongue circulation time" was fifteen seconds. This compares favorably with the average arm to arm circulation time found in fifty-three normal individuals by Blumgart, using the radioactive method.
4. A crude pulmonary circulation time was obtained by injecting decholin into the external jugular vein. The average jugular to tongue circulation time was eleven and eight tenths seconds in twelve normal individuals.
5. Since the test depends on a subjective response, it was compared in fifteen normal individuals with the sodium cyanide method which produces an objective reaction. Comparison demonstrated that the subjective nature of the response to decholin in normal individuals does not militate against the accuracy of this test.
6. The circulation time was measured with decholin in various pathological states, including hyperthyroidism, secondary anemia, arterial hypertension, pulmonary emphysema, and congestive failure. The results were in accord with those obtained by Blumgart with the more accurate radioactive method.
7. In the amounts utilized decholin produced no untoward reactions in over 150 patients.
8. The decholin test recommends itself to the clinician by its simplicity, its safety, and the readiness with which it can be repeated and used at the bedside or in the office.
9. The measurement of the velocity of blood flow is an important clinical measurement and should be used widely by the clinician. The simplicity and safety of the decholin method should enable the clinician to make use of the measurement of the velocity of blood flow as an additional aid in the diagnosis and prognosis of diseases associated with dyspnea and edema.

We express our gratitude to Mr. Paul de Haen of Riedel-de Haen, Inc. for generously supplying the "Decholin" used in this study.

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CANCER OF THE MALE GENERATIVE ORGANS

BY FREDERICK L. HOFFMAN, LL.D.*

MOST of the authorities on cancer of the male generative organs take occasion to emphasize the rarity of this affection in this country and Europe. Almost without exception the conclusions are based on fragmentary data or personal experience, insufficient for the purpose. The latest authority, Dr. Arthur Purdy Stout, in an elaborate treatise on "Human Cancer" observes with reference to cancer of the penis and male urethra that, "Cancer of the penis is rare in the Americas and in Europe. One has only to refer to the small number of cases reported to feel sure of this. Moreover, if its clinical incidence is rare, it is even rarer as a cause of death and it is seldom found among autopsy statistics. Foderl (1926) reports only 40 cases in twenty-four years in Hochenegg's Clinic; Squirru (1924) encountered only 4 cases of penile cancer among 1000 cancers in the Argentine; Shore (1928) records only 4 cancers

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of the penis among 1000 miscellaneous cancers seen at the St. Luke's Hospital, New York, and there was only 1 cancer of the penis among 1862 cancers studied at the Presbyterian Hospital, New York."

For some years past I have made efforts to collect more satisfactory evidence for cancer of the penis, particularly as a cause of death. Through the kindness of the Director of the Division of Vital Statistics of the United States Census Office, I have been furnished with transcripts of death certificates representing the entire mortality from cancer of the penis for a period of years. During 1926-1930, the number of deaths from cancer of the penis recorded by the federal census office was 935, dying at an average age of 63 years.

For 1926, however, the returns were not entirely complete so that the actual number is probably nearer one thousand. The average age at death in 1926 was 66.5 years, in 1927, 62.6; in 1928, 62.9; in 1929, 62.5 and in 1930, 62.5 years.

In 1930 there were 225 deaths from cancer of the penis distributed according to marital condition as follows: married 118; single 32; widowed 65; divorced 4; unknown 6. By race, there were 173 white persons and 51 colored, and 1 unknown.

The occupational distribution reflects nothing very unusual, most of the men having been laborers, or 56; farmers 50; sailors, only 1; commercial employees and clerks 20; professional 9; machinists and mechanists 7; textile 2; painters 7; carpenters 10; shoemakers 3; tailors 1; bakers 2; butchers 1; miners 2; chauffeurs and teamsters 10; miscellaneous 11 and unknown and retired 33. As far as it is possible to judge there is no occupational predisposition to cancer of the penis to be derived from the available statistical information.

The range in ages in 1930 was from 25 to 99 years. Thirty-one were ages 80 and over. The average age of whites was 64.5 and of colored 56.0, and for both races combined, 62.5 years. The actual number of deaths from cancer of the penis for each year for which complete returns are available is 220 in 1927; 201 in 1928; 209 in 1929 and 225 in 1930. Considering the immense mortality from cancer, all forms, it is probably correct to say that relatively cancer of the penis is rather a rare disease in this country but it is of sufficient numerical importance to demand attention. Compared with cancer of the scrotum, cancer of the penis is relatively of much more importance.

For the years 1926-1930, with incomplete returns for the first year, the total number of deaths from cancer of the scrotum was recorded as 167, at an average of 69.6 years. The deaths by single years have been as follows: 1926, 32; 1927, 29; 1928, 31; 1929, 41; 1930, 34. The average age at death was 58.3 years in 1926; 60.8 in 1927; 59.5 in 1928; 57.9 in 1929 and 61.8 in 1930. The range in ages during 1930 was 15 to 92 years. The only striking occupational exception occurs in the case of the textile trade which includes a number of deaths from cancer of the scrotum due unquestionably to occupational conditions. These cases I have discussed in my paper on Mule-Spinners' Cancer contributed to the proceedings of the American Medical Association, January, 1928.

The foregoing observations are based on the reëxamination of original death certificates of cancer of the penis and scrotum. For cancer of the testes, I have no detailed information but I give the returns as published by the Census Office for the period 1924-1929: 1924, 210; 1925, 227; 1926, 225; 1927, 206; 1928, 284 and 1929, 281; a total of 1433 deaths in six years.

To illustrate the precarious nature of hospital statistics utilized for the present purpose, I give the returns for the Huntington Memorial Hospital for the period 1921-1932. During these eleven years the Huntington Memorial Hospital treated 57 cases of cancer of the penis, 3 cases of cancer of the seminalis vesicula, 6 cases of cancer of the scrotum and only 3 cases of cancer of the testes.

During the six years ending with 1930 the State Institute for the Study of Malignant Diseases in Buffalo, treated 34 cases of cancer of the penis, 4 cases of cancer of the scrotum and 12 cases of cancer of the testes.

Compared or rather contrasted with the mortality from cancer of the prostate, it is correct to say that cancers of the penis and scrotum are relatively rare but collectively represent quite a substantial number of patients of whom the large majority come for treatment when the disease has advanced to a practically hopeless condition. It requires only to be pointed out in this connection that the actual mortality from cancer of the prostate was 2,558 in the United States registration area in 1924, and 4,359 in 1929. The statistics for 1930 have not yet been published.

Another statement frequently made in connection with the foregoing is that cancer of the penis is more common in Europe than in this country. This is no doubt correct for the actual number of deaths from cancer of the penis in 1930, for example, was 162 in England and Wales. Based on the estimated male population the death rate from cancer of the penis in England and Wales was 6.6 per million during the period 1911-1920; 6.9 in 1926; 6.4 in 1927; 6.1 in 1928; 5.7 in 1929 and 6.3 in 1930. The rate for cancer of the scrotum has changed from 2.4 per million in 1911-1920 to 2.7 in 1926; 3.0 in 1927; 3.1 in 1928; 2.7 in 1929 and 2.3 in 1930. During the eight years ending with 1930 there have been 1,233 deaths from cancer of the penis and 544 deaths from cancer of the scrotum. The latter affection has received considerable attention of recent years on account of the unquestionable relation of this affection to mule-

spinners' occupation, largely centralized in the Lancashire area.

In Scotland, cancers of the testes, penis and scrotum are combined in the returns and are not strictly comparable with those for England and Wales or for this country. The actual number of deaths from these three affections during the period 1921-1930 has been 290 or respectively 131 during the first five years and 159 during the last.

Aside from the foregoing I have detailed statistics for a number of American and Canadian communities or sections from which I have obtained actual transcripts of death certificates for a period of years. Of 15,644 male deaths from all forms of cancer, only 55 were due to cancer of the penis or 0.35%, while 73 were due to cancer of the testes or 0.47%. Unfortunately deaths from cancer of the scrotum are not separately classified. Very few localities or countries of the world classify separately deaths from cancer of the male generative organs so that the foregoing represents most of the available statistical evidence on the subject.

A recent study of 60 cases of cancer of the penis by Dr. Bernard F. Schreiner of the State Institute for the Study of Malignant Diseases, Buffalo, N. Y., adds some very useful information as regards treatment. Another extremely interesting and useful contribution to the study of penile cancer is by Dr. A. L. Wolbarst of New York, contributed to the *Lancet* under date of January 16, 1932 with particular reference to the non-occurrence of penile cancers in the Jewish population. I have been able to confirm this conclusion through my extended study of cancer in the Jewish population of Amsterdam, as well as among the Jewish population of Montreal. In neither of these investigations did I find a single death from cancer of the penis, the non-occurrence of which is properly ascribed to the practice of circumcision. The same conclusion also applies to the Mohammedan population although certain exceptions have been noted. I am not familiar with any useful data at present available for or against this contention.

But much more impressive is the evidence regarding the extraordinary frequency of penile cancer in certain populations of the Far East. Stout observes in this connection that, "The story is quite different elsewhere, especially in the Asiatic countries. Maxwell (1928) found that the breast, the penis and the uterus were the organs most frequently affected by cancer among the Chinese in the order named; de Vogel (1928) found 5 cases among 61 malignant neoplasms among the Chinese tin miners on the island of Billiton, Dutch East Indies; Sitsen (1928) found that it is very frequent among the Malays of the Dutch East Indies."

Hastings Gilford, in his monumental treatise on "Tumors and Cancers" (1925) points out that Marmaduke Shield, in investigating the subject, found that in only a few instances did cancer of the penis originate in men apart from phimosis, and that "The evidence that this form of cancer is much more common among those who do not observe the rite of circumcision than among those who do is overwhelming. Seldom met with among Jews and Mohammedans it is comparatively common among the Hindus and Japanese. That cancer of the penis is not a common disease in Great Britain (100 among 9,685 cancers in men in 1900) and is still more uncommon in the United States, suggests that uncleanliness is the main cause. Yet according to Rodman and other observers cancer of this same part is rare among Negroes, who are peculiarly liable to phimosis. In 1900 there was not a single case of cancer of the penis reported among Negroes in the United States."

How far this statement is correct I am not prepared to say for my own investigations among Negroes in Virginia and New Orleans have been rather inconclusive. In any event I found that among 786 white male cancer deaths in New Orleans, there were only 4 deaths from cancer of the penis or 0.51%, while among 196 deaths from cancer among male Negroes, there were 3 deaths from cancer of the penis or 1.53%. Personally I am inclined to think that the disease is more common among Negroes than among whites.

But overwhelming is the evidence for the Far East. I have tabulated the details of pathological specimens examined by the Institute for Medical Research, Kuala Lumpur, Federated Malay States, for the period 1926-1931 with the following results. For all races combined, there were 517 specimens of males, of which 58 were from cancer of the penis or 11.2%. In addition there were 6 specimens from cancer of the testicles or 1.2%. There were apparently no specimens from cancer of the prostate. Among the Chinese only there were 340 specimens from cancer, all forms, of which 38 or 11.2% were cancer of the penis and 1 specimen from cancer of the testicle or 0.3%. Among the East Indians, there were 120 specimens from cancer, all forms, among the male population, and of this number 20 or 16.7% were cancer of the penis, and 4 cancer of the testicles or 3.3%.

The foregoing evidence, therefore, is overwhelming that penile cancer is extremely frequent among the Chinese and East Indians in the Federated Malay States, while evidence to the same purpose is obtainable for China. In an analysis by Dr. J. L. Maxwell, in the *Chinese Medical Journal* for 1928, is a tabulation in great detail of cases of cancer in Chinese Hospitals, numbering 1,133 for both sexes combined and of this number 159 or 14.03% were cancer of

the penis. There were 6 cases of cancer of the testes equivalent to a percentage of 0.53. But unfortunately the separation of the data is not by sex so that the exact proportion of the male population cannot be calculated. It may also be pointed out in this connection that Dr. Maxwell compares the Chinese hospital returns with the mortality returns in similar detail for the city of Chicago, derived from one of my reports. Such a comparison, of course, is utterly fallacious since hospital returns of patients cannot be compared with mortality returns which invariably reflect a different situation. But the data are fully convincing to the effect that cancer of the penis in China is relatively very common among the Chinese population and this is supported by data furnished by Sir Leonard Rogers, also unfortunately for both sexes combined. He gives returns for 689 deaths from all forms of malignant disease verified by autopsy for the Calcutta area of which 18 were cancer of the penis and 240 were epithelial squamous tumors. This, of course, is a much lower rate of incidence than that revealed for the Federated Malay States, but much higher than would be the case in European or American localities or sections. Since the data are not given separately for the male and female populations no exact rate of incidence can be calculated.

I had expected to find a very high rate of incidence of cancer of the penis in the Philippines but a careful examination of the original death certificates for four years reveals a surprisingly small figure for cancer of the penis and for cancer of the remainder of the male generative organs. For the city of Manila, I have returns for 167 male deaths from cancer for the two years 1926 and 1927, of which only one was a death from cancer of the penis and one from cancer of the scrotum or respectively 0.6% for each type. Cancer of the prostate also is very uncommon, there being only one death from this affection. In the earlier returns for 1924 and 1925 I find 137 deaths of males from all forms of cancer, of which none was attributed to cancer of the penis, only one to cancer of the testicles and 3 to cancer of the prostate. It is thus shown that the unusual incidence of cancer of the penis is highly localized and that the conclusion is based on general data especially as derived from hospital returns which have to be dealt with in an extremely cautious manner.

I cannot omit, however, in this connection to draw attention to the admirable discussion of the whole subject of penile cancer by Dr. Wolbarst (1932) which includes a table for India which provides data of the utmost value. Wolbarst presents returns for Indian hospitals 1925-1930 representing 7,692 cases for all forms of cancer in the male population of which 1200 were penile cancers or 15.60%. He gives returns separately for Hindus and Mohammedans, there having been 1,169 cases of penile cancers among the Hindus and only 24 among the Mohammedans among whom circumcision is the general rule. He combines these official figures with those of Sutherland and Megaw, deriving a total of 8,484 cancers in men. Of these, 1,336 were penile cancers, of which 1.303 occurred in Hindus or 97.5%, and only 2% in Mohammedans.

He points out that the proportion of Mohammedan patients treated in hospitals under review was 21% and that therefore if penile cancer occurred with equal frequency in both races, the number of cases reported in Mohammedans should approximate 254 instead of the 24 cases actually reported. There are many other very interesting observations in Wolbarst's paper which makes it one of the most useful and interesting on the subject. He concludes by quoting the returns for the United States 1927-1929 when out of 144,206 male cancer deaths, 654 were penile or 0.45%. The proportion for England and Wales during the same period was 0.61%. He also quotes the experience of the Veterans' Bureau 1923-1925, with a mortality ratio for penile cancer of 2.4% out of a total of 124 deaths from cancer, all forms. He also quotes Lieut. Commander Parsons of the U. S. Navy in Haiti to the effect that penile cancer in Haiti is extremely common. This would support some of my own conclusions regarding penile cancer in the American Negro.

THE OPERATIVE CURABILITY OF CARCINOMA OF THE STOMACH*

BY LANGDON PARSONS, M.D.†

IN current discussions of the treatment of carcinoma of the stomach one cannot help but be depressed with the attitude of hopelessness shared by internists and surgeons alike. It is true that carcinoma of the stomach is the most common cause of death from cancer in men and only outranked in women by carcinoma of the breast and uterus. Ten thousand deaths occur from this disease annually in the British Isles alone. An analysis of the so-called cures following surgical treatment of carcinoma of the stomach however, are all the more discouraging when we consider that we are dealing with an organ

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NOTE: This analysis was undertaken at the suggestion and with the helpful criticism of Dr. Lincoln Davis.

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readily approached and treated by surgical measures.

Bloodgood[1] analyzed all the cases of carcinoma of the stomach entering the Johns Hopkins Hospital up to the year 1915 and found but one five-year cure. From all sources in the literature in 1913 there were but three five-year cures, and 75 per cent of the cases were considered inoperable.

Surgical technique generally shows greater perfection. We have at our disposal greater and more accurate means of arriving at an accurate diagnosis. Certainly x-ray diagnosis has definitely improved. Yet fifty per cent of the cases of carcinoma of the stomach are found to be inoperable and when resection is possible the chance of cure appears less than thirty per cent. Balfour[2] suggests the answer in a recent discussion before the American College of Surgeons, in remarking that while the laity are more hopeful due to the intensive educational campaign putting forth the facts in regard to cancer, a stalemate is reached when the "pessimistic attitude of the physician provides little encouragement for the only program which offers any help, namely the surgical removal of the local growth and regional lymph nodes." It was with this pessimistic attitude in mind that the analysis of 233 cases of carcinoma of the stomach was undertaken hoping to show that carcinoma of the pyloric half of the stomach belongs to the curable type of cancer.

During the five year period 1922-1926, 233 cases of carcinoma of the stomach entered the Massachusetts General Hospital on the medical or surgical wards, about 12 per cent of the total number of cancer admissions. Of these, 139 cases came to operation while the balance were either regarded as inoperable or were offered operation and refused. The average length of life of the group, where no operation was performed, was six months. The longest duration of life was one year and eight months in a case of carcinoma high up on the cardia without obstructive symptoms.

SURGICAL CASES—139

Explorations (only)	63 or 45 per cent	with an operative mortality of 27 per cent.
Anterior Gastroenterostomy	18 or 12.9 per cent	" " " " " " 22 per cent.
Posterior Gastroenterostomy	21 or 15.2 per cent	" " " " " " 33.3 per cent.
Gastric Resections	37 or 26.6 per cent	" " " " " " 38 per cent.

The average length of life of patients surviving operations was 15.4 months. The patients who had simple explorations lived on an average three months. The patients in the anterior gastroenterostomy and posterior gastroenterostomy groups survived on an average eight months. The average length of life in the group in which resection was performed was 39 months.

Obviously, it is only in the group of cases where gastric resection was done that hope of cure can be found. The mortality from resection of whatever type is high, 38 per cent, but this may be explained, in part at least, by the fact that the operations were performed by nine different general surgeons. The average age of the patients in the cases with gastric resection was 51 years. The youngest patient was 35 years of age, the oldest 66 years. Nine patients (24.3 per cent) lived five years after the operation. Excluding the immediate operative mortality of 38 per cent, the five-year survival percentage mounts to 38 per cent. At the time of the operation three of the cases had metastases in the lymph nodes of the lesser omentum proved by microscopic examination. Three of the cases died after five years had elapsed, two of known recurrence of the disease. The remaining six are alive and well today, one 9½ years, two 9 years, one 8 years, one 6½ years and one 6 years following operation. All but one have been examined and x-rayed within three months and no evidence of disease found. The sixth case writes from Finland that she is alive and well enough to outlive the last of three husbands acquired since her operation nine years ago. All but one of the cases are doing heavy work. The one exception at the age of 73 years was recently readmitted to the hospital with a dislocated shoulder*.

On the basis of the results in this series of cases, a patient with carcinoma of the stomach may be advised that if he refuses operation he will live on an average six months. Exploration or palliative procedures offer him no hope of cure and an average life expectancy of three months or eight months respectively. If resection can be carried out he has a 62 per cent chance of surviving the operation. If he survives he may then expect a 38 per cent chance of living five years or more and a 56 per cent chance of living three years or more.

The reasons most commonly advanced as to why operation should not be considered, the exploration abandoned or palliative procedures other than resection instituted were as follows:

(1) Presence of a palpable mass.

(2) Fixation of the stomach or mass as noted by x-ray or at operation.

*Within the past three weeks one of the five-year survival group reëntered the hospital deeply jaundiced. Exploration revealed a stomach without evidence of disease six years after operation. The liver was studded with nodules. The pancreas was thickened and indurated. A cholecystjejunostomy was done. Later the patient came to postmortem. No evidence of recurrence of the original disease could be found. The patient died of a primary carcinoma of the pancreas. A thorough review of multiple paraffin sections was done to exclude the possibility of a recurrence of the original gastric lesion. Another factor of interest was the appearance of the stomach (Billroth II), which did not present the physiological dilatation one might expect. This may well explain the frequent observation following a gastric resection that food does not distress, but that the patients cannot eat so much as they formerly did.

(3) Presence of an enlarged liver.
(4) Extension (apparent) of the disease to the regional lymph nodes.
(5) Possibility of ulcer rather than carcinoma.

PALPABLE MASS

The presence of a palpable mass was frequently regarded as a contraindication to resection based on the supposition that the actual presence of a mass presupposed a long-standing growth with resultant distant metastases. Yet in this series 29.3 per cent of the resected cases had a palpable mass. Of the five-year cures 44 per cent had a mass which could readily be felt preoperatively. Tumors are known to grow to a very large size before causing symptoms, while smaller lesions from their location at the pylorus early give rise to obstruction necessitating operative interference. The tumor mass in the cases in this series where gastric resection was the operative choice ranged from 2″-5″ in length. Balfour[2] notes that a large freely movable cancer is more likely to prove resectable than the cancer which cannot be palpated.

Walton[3] expresses the situation admirably when he observes that the mere presence of a tumor is no contraindication to operation, nor is its size or apparent mobility.

FIXATION OF THE MASS

The internist and surgeon alike are prone to take the roentgenologist's opinion as to the possibility of resecting the tumor mass. If he reports fixation of the lesion, exploration is either not considered or is approached with little hope of any permanent cure. In a large number of cases in this series fixation of the tumor was noted on roentgenological examination and the tumor regarded as inoperable, yet at operation resection proved possible and the fixation was found to be more apparent than real. In every instance where anterior gastroenterostomy was performed because of the adherence of the growth to the underlying pancreas, no note was made of fixation preoperatively by the roentgenologist. Bloodgood[4] appears to be correct in pointing out the danger of concluding on the basis of x-ray studies alone that a lesion in the stomach is inoperable.

Even at operation fixation of the tumor mass is often given as good and sufficient reason why resection of the stomach should not be undertaken, particularly when there is adherence of the neoplasm to the underlying pancreas. In 13 per cent of the cases in which exploration alone was done and in 22 per cent of the cases where a palliative operation was performed, adherence to the pancreas was given as the main consideration in determining the type of operation. Immobility of course adds to the technical difficulty of the resection, yet we find that 26.5 per cent of the cases having a gastric resection were adherent to the pancreas. One of these is alive and well nine and one half years after operation. Balfour[2] adds a practical note that complete relaxation under anesthesia is necessary before the degree of immobility can be adequately determined. Many growths appear fixed with the patient straining under light anesthesia that with relaxation prove readily accessible.

LYMPH NODES

The presence of nodes high up on the lesser curvature, suprapyloric, subpyloric or along the greater curvature was advanced as reason for resting content with a palliative procedure. This was noted in seven per cent of the explored cases and in 14 per cent of the palliative cases. Operable—"yes"; curable—"no"; occurred many times as an operative note. Yet it is impossible to know at the time of operation whether the nodes are involved. In many instances the surgeon definitely pointed out gross evidence of malignancy in the lymph nodes of the omentum only to be surprised when the pathologist could find no evidence to substantiate the gross diagnosis. Conversely, the surgeon noted grossly involved lymph nodes at the time of operation in 47 per cent of the cases whereas the pathologist found microscopic malignancy in 59 per cent. Balfour and Walton add their confirmation that enlargement of the regional lymph nodes does not necessarily mean malignant involvement.

Should a case be considered inoperable when the lymph node involvement is so extensive as to make their complete removal impossible? A large number, 59 per cent, of the cases in which resection was done in this series showed malignant cells in the lymph nodes on microscopic examination of the sections. Moreover, 25 per cent of the 59 per cent occurred in the three and five year survival group. Balfour[2] feels that the same observation that is repeatedly made in dealing with cancer of the breast, rectum and pelvic organs likewise applies to cancer of the stomach, namely the removal of the primary growth and of the immediately adjacent lymphatic structures may bring about a prolonged arrest of the disease even if involved lymph nodes are left. The remaining nodes in such cases seem to act as a barrier to further dissemination of the disease. The fact that seven of the nine five year survivals had metastases in the lymph nodes at the time of operation, all showing a high degree of malignancy as determined microscopically, tends to support Balfour's contention.

Bloodgood[4] emphasizes the fact that the removal of a node near the stomach or in the omentum for immediate microscopic study does not exclude cancer if there is no metastasis, nor does it contraindicate resection if there is.

LIVER METASTASES

Surgeons and internists are apt to take the point of view that cancer of the stomach metas-

tasizes to the liver early in its course, thereby nullifying all hope of cure by radical gastrectomy. Cheever[5], however, found in reviewing a series of cases in which the disease was so far advanced as to lead to death without operative interference, that postmortem examination showed no liver metastases in 22.8 per cent. The disease, then, may run to a terminal issue without showing liver involvement. Warwick[6] reported liver metastases in 38 per cent from 176 autopsies of cancer of the stomach. The liver was definitely the site of metastases in 49 per cent of this group of cases.

Even when there was actual malignant involvement of the liver it is apparent from an analysis of the follow-up reports in this series that the patient was more comfortable after resection than when a palliative operation was performed, because of the apparent hopelessness of the situation. This may be due to the removal of the foul ulcerating lesion and the freedom from the constant gnawing pain in the epigastrium. With this contention Balfour agrees, being impressed with the number of patients who had irremovable metastatic growths, who lived in comparative comfort two, three and five years following palliative resection. In many cases of this series where resection was carried out obviously for palliative reasons alone, the patient outlived in comfort by several years the prognosis given at the time of the operation. More evidence is thereby added to the clinical impression that in dealing with malignant disease the removal of the local lesion has some inhibiting effect on the growth of remote metastases. Bloodgood and others agree that "resection of the stomach where possible offers the patient more comfort and perhaps lengthens lives even when there are metastases to the regional nodes of liver."

ULCER VS. CARCINOMA

Every surgeon interested in gastro-intestinal surgery has been faced with the decision as to whether a lesion in the pyloric half of the stomach represents malignant or chronic gastric ulcer. In seven per cent of the cases in this series gastroenterostomy was done because of the opinion that the tumor mass was an ulcer rather than carcinoma. MacCarty[7], Stewart[8], Scott[9], Masson[10], Duggan[11] and others seem to agree that a clinically diagnosed chronic gastric ulcer in the pyloric area will prove to be cancer in 10 to 20 per cent in patients presenting ulcer symptoms that have existed over a period of months or years. Aschoff[12], a strong opponent of the "carcinoma-from ulcer" theory, expressed surprise at the large number of resected specimens grossly resembling ordinary round ulcer which on microscopic examination proved to be cancer. It is quite possible that these lesions may be cancer from the beginning, but the diagnosis clinically is impossible. MacCarty[13] states that from an experience with more than 1400 gastric specimens "the association is so frequent that if I had a chronic gastric ulcer I should always consider the possibility of a cancer being present and I know of no clinical or laboratory methods by which the differentiation can be made."

The Mayo Clinic[14] in tracing 1280 cases where posterior gastroenterostomy was done for either gastric or duodenal ulcer found 195 deaths three and one-half years after the operation; 75 of these 195 cases were proved to have had carcinoma. The duodenal ulcer group followed the normal life expectancy for unoperated cases of the same age, whereas the gastric ulcer series showed three times as many deaths. Circumstantial evidence, in view of the 75 deaths proved to have been due to carcinoma, would suggest that either the cases were carcinoma from the beginning or that carcinoma developed in a chronic ulcer. In our small series there were eight cases where gastroenterostomy was done for ulcer in which carcinoma was proved at a later date. The mortality in resection for gastric ulcer is variously reported as six per cent whereas in resection for carcinoma the figures run from 10 per cent to 38 per cent. It would seem that even if there is reason to believe that a lesion is ulcer rather than cancer, resection should be elected rather than posterior gastroenterostomy.

SYMPTOMS

Sir James MacKenzie[16] has said if we are to make any progress in the medicine of the future it will be through the interpretation of early symptoms. The earliest symptoms were those of fullness in the epigastrium with discomfort and burning. Lack of appetite with a distinct distaste for food occurred in 29 per cent of the cases. Later, nausea and vomiting appear but usually at a more advanced stage in the disease, often associated with emaciation and the presence of a palpable tumor. It has been suggested, however, that a small tumor located at the pylorus may give rise early in its course to obstructive symptoms. The pain and discomfort were variable factors. Two patients had absolutely no gastrointestinal symptoms.

It was of interest to note the frequency and prominence with which constipation appeared in the list of symptoms. Thirty-eight per cent noted a change in bowel habit. Some of these cases were referred to the hospital with a diagnosis of carcinoma of the colon.

It is surprising how little significance the patient places on loss of weight. The average weight loss from the time of the first observed symptom was one and one-quarter pounds a week, with an average loss of two pounds in the five-year group.

In a large number of cases of carcinoma of the stomach, the lesion is inoperable when the symptoms first manifest themselves. Walton[3] placed it as high as 65 per cent and Dwyer[17] at 62 per cent. We might expect to find that in the

inoperable groups the symptoms would be present for years in varying degrees of severity with little attention being paid to them, until the patient no longer is able to continue at his work, entering the hospital only to be declared hopelessly inoperable. Yet we find that 27.5 per cent of the inoperable cases had symptoms for only one to three months while 47 per cent of the patients had noted their symptoms for six months previous to the time of entry in the hospital. In the resected group, conversely, we might expect a shorter duration of symptoms and a relatively early apprehension of the disease, consequently a more favorable case for resection, yet we find that 48.3 per cent of the total had symptoms from one to five years.

From these observations the assumption may be made that in the group with the extensive malignancy and relatively short duration of symptoms either the neoplasm was of a high degree of malignancy or the resistance of the patient was definitely lowered.

On the other hand in the groups of patients with the longer duration of symptoms it is fair to assume that the growth of low malignancy has either taken on a higher degree of activity or again that the defensive factors have been reduced. The possibility of the development of malignant changes on the basis of previous known pathology such as an ulcer can be overlooked. But whether you believe that cancer develops from ulcer or is a neoplasm from inception it would appear that at some time in the duration of symptomatology the lesion was under control of the body defense. Gastric resection at such a time might well be expected to lead to a permanent cure.

Thus Alvarez[18], Walton[19], Scott[9], and others contend that all patients above the age of thirty-five complaining of chronic epigastric distress or a sudden change in bowel habit should be regarded as cancer suspects until the latter is ruled out.

LABORATORY FINDINGS

The absence of free hydrochloric acid has come to be associated with the diagnosis of carcinoma of the stomach, yet its presence was noted in concentration ranging from 2 to 18 in 28 per cent of the cases. It was an interesting observation that the cases having an absence of free hydrochloric acid tended to fall into the inoperable group with an average duration of symptoms of six to seven months and were frequently found to have a filling defect in the upper portion of the stomach as shown by x-ray.

Where free hydrochloric acid was present the filling defect occurred at the pylorus in 83 per cent of the cases associated with a history of chronic epigastric distress over a period of years.

A mild anemia was invariably present but in only two cases did the red blood corpuscles fall below three million with a hemoglobin of less than 50 per cent. The patients appeared cachectic out of proportion to the anemia shown. Bleeding from the gastro-intestinal tract does not explain the anemia, for in 20 per cent of the cases the guaiac test on the stools was negative.

X-RAYS

Certainly in the consideration of symptoms or physical findings there are few outstanding landmarks to point the way to an early diagnosis. Likewise the laboratory data are often misleading. X-ray appears to be our main hope. Cheever[5] has reported that a better than 90 per cent correct diagnosis can be made by a competent roentgenologist. Bloodgood was unable to find a single case where cancer had been overlooked after a gastro-intestinal x-ray examination. In this series, 12 per cent of the total cases were diagnosed as ulcer but this variation may in part be explained by the fact that the x-ray examination is done by men in training as roentgenologists. The greatest difficulty appears in the diagnosis of carcinoma high up on the cardia; rarely is it missed at the pylorus. Stewart's[8] analysis of 120 cases of carcinoma of the stomach showed that 67 per cent of the cancer cases occurred in the pylorus or pyloric one inch.

Hampton, of the Massachusetts General Hospital Roentgenology Department, contends that an ulcer found in the prepyloric area is malignant. As previously noted, eight cases in this small series where posterior gastroenterostomy was done for ulcer were later operated upon for carcinoma. In 64 per cent of the total cases it was noted that peristalsis was lacking or sluggish at the site of the filling defect. Fixation of the stomach was noted in ten per cent, yet we have noted elsewhere that it is dangerous to conclude through x-ray alone whether a lesion in the stomach is operable.

It would seem then, in view of the success of the roentgenologist in making a correct diagnosis of carcinoma of the stomach, that our hope of finding cases more suitable for resection at least in the group presenting symptoms of one to five years' duration might well be realized. Yet in this connection, Alvarez[20] has written an interesting paper on how often physicians make a diagnosis of cancer of the stomach in themselves and he finds it hard to understand how a physician with ulcer symptoms could let himself go without a gastro-intestinal x-ray.

SUGGESTED MANAGEMENT OF A CASE

A patient, then above the age of thirty-five years with such gastric complaints as belching, distress or discomfort after eating, loss of appetite, weakness or constipation coming on as a definite change in digestive habit, should be regarded as a cancer suspect and receive a gastro-intestinal x-ray examination at the hands of

a competent roentgenologist. If the diagnosis of neoplasm is made, immediate operation should be advised. After proper preliminary preoperative care as regards transfusion, lavage and intravenous glucose, gastric resection with removal of the adjacent lymph nodes should be carried out if technically possible. Where complete removal is impossible, a resection for palliative reasons alone will give the patient more comfort and prolong life to a greater extent than gastroenterostomy.

On the other hand, should the roentgenologist report an ulcer other than in the prepyloric area, thorough and rigid medical treatment should be instituted. Wheeler[19] believes that if there is no gain in weight, if there is a failure to maintain weight, or if there is a change in character of the symptoms during efficient medical treatment, malignant disease should be suggested and exploration justified.

SUMMARY

From the consideration of this group of two hundred and thirty-three cases it would appear (1) that our future success in the diagnosis and treatment of carcinoma of the stomach lies as much in the education of the profession as of the laity; (2) that after a consideration of symptoms we have the means at hand of making an early and accurate diagnosis by x-ray examination; (3) that dependent on the above factors, appreciably more than fifty per cent of the cases can be considered operable; (4) that gastric resection is the operation of choice both from the point of view of comfort and of longevity.

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FUNCTIONAL BUNDLE BRANCH BLOCK

BY F. B. CARR, M.D.*

THE electrocardiograph has proved its worth in a great many ways over the past few years. Its clarification of the mechanism involved in the production of the various arrhythmias and disorders of the heart beat has been not only of great theoretical interest but likewise of important practical value. The prognostic significance of many types of tracings has not as yet been determined. It will take further years of careful observation to derive the utmost value from this method of clinical investigation.

The electrocardiographic diagnosis of defective intraventricular conduction implies an increase in the time required for the propagation, through the ventricles, of the excitation wave. The QRS complex requires 0.10 seconds or more for its accomplishment. When one of the two main ventricular conduction bundles fails to function, a condition of bundle branch block is said to exist. Ordinarily the occurrence of this condition renders prognosis serious by reason of underlying pathology sufficiently extensive to cause this conduction damage.

The diagnosis of defective intraventricular conduction requires the use of the electrocardiograph. But here lies a pitfall. There is increasing evidence to show that not only an organic obstacle may oppose the passage of the impulse, but there may be functional interference causing only a transient conduction defect. An increased vagal tone may inhibit the transmission of impulses along one or the other bundle. Ample evidence of the existence of such a condition may be found in the paper of Wolff, Parkinson and White[1]. Functional bundle branch block occurred in eleven young and healthy persons. These incidences of block were evidently caused by increased vagal control inasmuch as they were prone to disappear when exercise reduced the influence of this nerve.

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Willius and Keith[2] have shown that the accumulation of so-called fatigue products in the region of the conducting bundles may initiate a functional block of this nature. The case to be presented in this paper falls into this latter category of functional block due to the presence of fatigue products. It is of interest because we were able to cause the disappearance of the block by vagal stimulation. To my knowledge only five other similar cases have been reported in the literature.

CASE REPORT. The patient is a fifty-two year old man, white, married, and unemployed. His first admission to the hospital was in October, 1930, at which time he had a surgical repair of a right inguinal hernia, under ether anesthesia. His heart was reported to be normal at this time, though no electrocardiogram was taken, and he made an uneventful convalescence. The day before his second admission, May 22, 1932, he commenced to have difficulty in breathing. Dyspnea became progressively worse until his local doctor advised that he come to the hospital. Aside from the dyspnea he complained of a small amount of pain about the left nipple. This, however, was not a very prominent feature.

The past history was not helpful. He had always been well. He could remember having none of the childhood diseases. He had never had any symptoms indicating cardiac disease except that two months before admission he engaged in a fistic battle, following which he noticed a sharp pain extending from about the left nipple through into his back. This lasted most of the night, but was gone in the morning.

Physical Examination showed the patient sitting up in bed breathing rapidly and showing some cyanosis of the lips and extremities. The pulse was 100 and regular. There were many coarse crepitant râles in both lungs increasing toward the bases. The heart did not seem enlarged to clinical examination. The sounds were not easily heard and no murmurs were recorded. The temperature was 98.8 degrees and the respirations were about 30. Otherwise physical examination was not abnormal.

Laboratory Findings. Repeated urine examinations were normal. The white blood cell count was 8,400 on admission and 9,400 on the fourth day. The red blood cell count was 5,200,000. Wassermann and Kahn tests were negative.

Subsequent Course. The blood pressure was 92/70 on admission, increasing to 100/60 on the third day. The temperature remained between 99 and 100 for eight days when it dropped to normal. There was gradual diminution in the number of râles and the patient was allowed out of bed on the nineteenth day. Treatment consisted of morphine and digitalis. The diagnosis was arteriosclerotic heart disease with left heart failure, although coronary thrombosis was not definitely ruled out. To the present time the patient has been feeling well, with no symptoms referable to the heart.

Electrocardiograms. Figure 1 shows the three leads taken on his admission to the Hospital, May 23, 1932. This shows a typical left bundle branch block

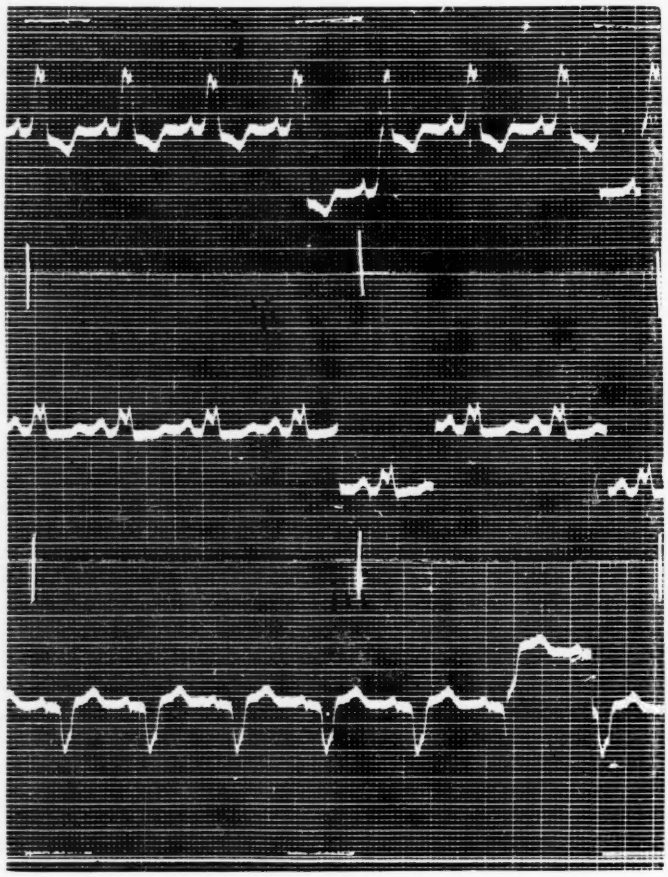

FIG. 1. Leads 1, 2 and 3, taken on admission to the Hospital, showing a typical left bundle branch block.

(new nomenclature). The QRS conduction time is 0.12 seconds; the PR interval is about 0.16 seconds. At the time this tracing was taken we felt that this was an example of a bundle branch block with an organic background, perhaps following coronary sclerosis. Figure 2 shows the three leads taken on June 15, 1932 after twenty-three days of rest. The bundle branch block has disappeared. The PR interval remains 0.16 seconds; the QRS time is 0.06 seconds and there is a left axis deviation. The explanation of this change is probably twofold. The heart rate has dropped from 94 to 62 beats per minute, thus increasing the TP interval and thereby allowing increased opportunity for the disposal of the products of fatigue. Also the twenty-three days of rest has probably restored the heart to more normal function.

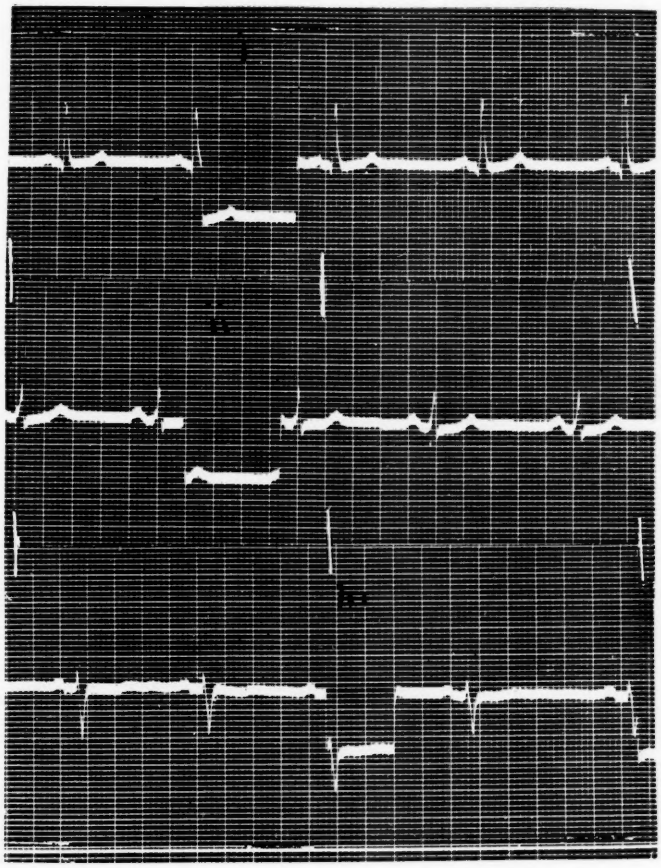

FIG. 2. Leads 1, 2 and 3, taken after 23 days of hospital rest, showing a return to normal intraventricular conduction time.

Figure 3 was taken about six weeks following his discharge from the hospital. The bundle branch block had returned in a control tracing practically identical with that of figure 1. Figure 3 (lead 2) shows the effect of deep inspiration in reducing the QRS complex from 0.12 seconds to 0.06 seconds, returning to 0.12 seconds with expiration.

Figure 4 shows a striking effect of pressure on the right carotid sheath (vagal stimulation). The bundle branch block is immediately supplanted by a condition of normal conduction.

Figure 5 shows the effect of vagal pressure during a follow-up visit about five months later. The patient had been feeling well and had been taking no medication. There is marked slowing of the ventricular rate but the QRS conduction time remains at 0.12 seconds.

DISCUSSION. A complete survey of the literature on functional (partial) bundle branch block is

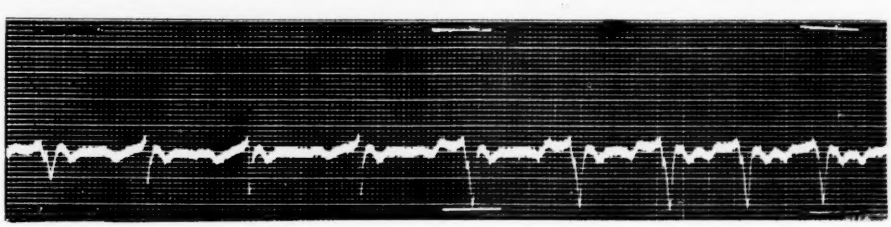

FIG. 3. Lead 2, taken 6 weeks after discharge from the hospital, showing the effect of deep inspiration in reducing the intraventricular conduction time from 0.12 seconds to 0.06 seconds.

found in a recent article by Herrmann and Ashman[3]. Among other cases they describe three instances of a type of bundle branch block which are quite similar to the case at hand.

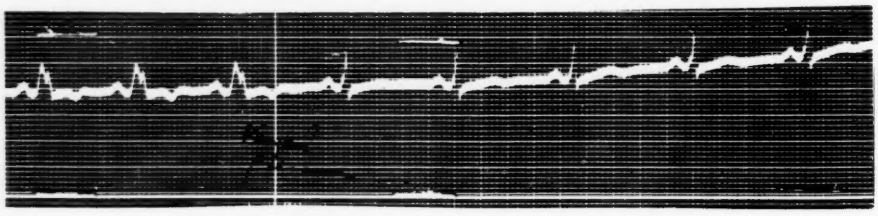

FIG. 4. Lead 2, showing the effect of right vagal pressure in reducing QRS from 0.12 seconds to 0.06 seconds. (White vertical line represents point of application of the pressure.)

Subsequently Elliot and Nuzum published an account of a case falling in the same category[4]. Sigler writes of a case of functional bundle branch block paradoxically relieved by vagal stimulation[5]. The case at hand then increases

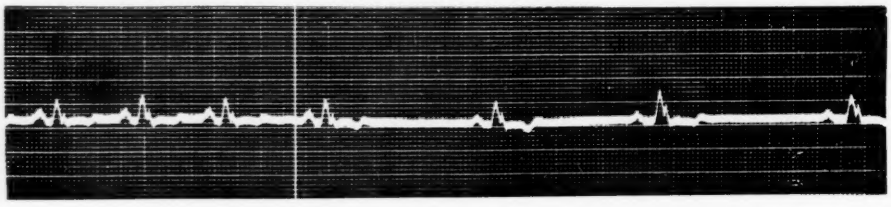

FIG. 5. Lead 2, 5 months later, when vagal pressure, though markedly reducing the rate, failed to restore normal intraventricular conduction time. (White vertical line represents point of application of the pressure.)

to a total of six the reported instances of bundle branch block abolished by vagal stimulation.

On the second follow-up visit of this patient, when the electrocardiogram showed the bundle branch block to be present, exercise failed to dispel the conduction defect. On the other hand we were surprised to find that pressure over the right vagus nerve, though reducing the ventricular rate to about thirty-seven beats per minute, did not restore normal intraventricular conduction as at the previous visit. This brings up two possibilities: either that the heart had reached such a state of fatigue generally that even such a marked prolongation of diastole was inadequate for the removal of the fatigue products from the neighborhood of the conduction bundle. The other possibility is that the hitherto functional bundle branch block has become organic in nature. We can speculate that behind the whole picture is a coronary sclerosis causing a localized interference with normal metabolic processes and evidencing itself in the functional conduction defect. Such a local situation would lend itself readily to the gradual formation of scar tissue, which, by incorporating the bundles, would act as an organic obstruction to the normal passage of impulses. If these assumptions are true, functional bundle branch block, when due to the accumulation of fatigue products cannot be classed as an innocuous condition. This in contrast to functional bundle branch block of vagal origin. Rather is the former condition to be interpreted in the light of certain underlying pathological processes which may come to have a grave prognostic significance for the patient.

SUMMARY. A case is reported in which the electrocardiogram showed a bundle branch block. This block could be terminated by vagal stimulation. Such a response could not be attained five months later. While this condition is called functional bundle branch block, it may have a graver significance than its name implies by reason of certain underlying pathological processes which are progressive in nature.

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STUDIES OF REPRODUCTION IN THE RAT*

1. The Effect of Changes in the Protein Upon Fertility, Pregnancy and Lactation

BY DONALD MACOMBER, M.D.†

INTRODUCTION

THE purpose of this and subsequent papers is to determine the effect of changes in diet upon reproduction in a quantitative way. For many elements of diet the groundwork has been done in showing the effect of changes in percentage intake. There remains to be worked out the complex inter-relationships. One of the difficulties of percentage feeding is that a change in the percentage of one element will cause changes in the percentages of all the others. We are dealing with many complex factors, hence the advisability of placing as many as possible on an accurate basis of total amounts ingested.

Of the various elements of which our diets are composed there is none more important, or about which there has been more discussion, than the protein constituent. The amount of protein on which nitrogen equilibrium can be maintained has been found to be relatively small[1], but most experts are agreed that the optimum is much higher. Protein has long been recognized as an important element in reproduction. In fact, the general impression has been that the amount of protein not only affects reproduction, but may be a limiting factor as it is in the growth of young. In 1922 Evans and Bishop[2] published some work on this point, but as the diets used contained only fat and protein, and their observations on the effects produced by changes in the percentage of protein upon reproduction were limited to the occurrence of estrus, no deductions can be drawn. In 1924 Simmonds[6] reported a large series of experiments showing the effect of changes in the percentage of protein upon reproduction and lactation. Diets ranging from 67 down through 57, 40, 35, 31, 21 to 9 per cent protein were used. In general, lactation was not adversely affected by percentages above 21 (the control) though many of the mothers eventually suffered kidney changes. Nine per cent was chosen for many of the diets since it was found that the proteins had to be of good quality for lactation to succeed. If the proteins were largely from animal sources lactation was found to be fairly normal even at this figure, but many failures occurred and the weights of the young at weaning were never so good. Furthermore, the mothers lost more weight. The main criticism of this work is that applicable to all percentage feeding experiments, namely, that the actual amounts ingested were not known.

There have been practically no other investigations reported on this subject until the past year when J. R. Slonaker[7] published four articles in the *American Journal of Physiology* on "The Effect of Different Per Cents of Protein in the Diet." Among other things the Effect of the Changes in Protein upon Reproduction was studied. Five diets were used so constituted that they had equal energy producing qualities combined with 10, 14, 18, 22 and 26 per cent protein respectively. The work was well done and the results are instructive. One criticism may be made as to the sources of the vitamins. The diet did not contain cod liver oil and very little butter (one per cent); yeast was 0.4 per cent; wheat germ 0.6 per cent. The author soon found that the diet was deficient in B, and this was corrected by adding fresh brewers' yeast, but A, D and E, while sufficient for reproduction, would seem to have been suboptimal. All the diets were made up from Diet I (protein 10 per cent) by adding meat scrap. This while increasing the protein could not help decreasing the per cent of vitamins, and the higher the per cent of protein, the lower must have been the per cent of these important elements. One cannot help wondering whether some of the ill effects noted with animals on these higher percentages may not have been due to slight deficiencies in these vitamins. In every instance the reproductive performance of rats on Diet II (protein 14 per cent) was superior to that of rats on other per cents. Fertility was higher and there was less sterility, the animals continued reproducing longer, the average number of litters was greater and the average number of young was higher; more young were brought to weaning. One strange fact was noted, namely, that the fertility of the males was greatly decreased below that of the females. Even on Diet II, where female fertility was 100 per cent, that of the males was only 87 per cent. Could this also be the result of a diet low in vitamin content? Deficiency in vitamin B is known to affect males, even causing testicular atrophy in extreme cases. This work of Slonaker, though important and carefully worked out, was also carried out by percentage feeding methods. No idea of actual protein intake can be obtained from the experiments as reported.

The present series of experiments was designed to be a quantitative investigation into the effect of changes in the amount of protein ingested upon reproduction, including fertility,

*The experimental work upon which this paper is based was carried out in the Research Laboratory of the Department of Obstetrics, Harvard Medical School. It was aided by grants from the Delamar Fund.

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pregnancy and lactation. Four diets were used which may be designated according to the percentage of protein they contained; I 20 per cent, II 16 per cent, III 10 per cent and IV 5 per cent. The protein used was commercial casein which is known to be adequate in all the amino acids necessary for growth. The other constituents of the diet were identical except for the fact that the per cent of starch was increased to compensate for the lessened per cent of protein. The control diet (Diet I—protein 20%+) was made up as follows—Casein 20 per cent; Salt Mixture IV (Osborne & Mendel[5]) 4 per cent; Starch (corn starch) 51 per cent; Wheat germ 5 per cent; Powdered yeast extract (Harris) 2 per cent; Cod liver oil (Patch) 2 per cent; Butter 16 per cent. Later a small amount of fresh lettuce was fed daily, also for a while 10 per cent wheat germ was used with a corresponding increase in the total protein to 21 per cent.*

The method of procedure was substantially as follows: At least ten adult females were kept on Diet I. Later ten more were added, making twenty in all which were used entirely as controls. Ten were put on Diet II and after a sufficient period of observation these rats were shifted to Diet III, and later to Diet IV. Finally these same ten rats were themselves placed on Diet I. Other parts of the experiment regarding energy metabolism, vitamin E, etc., will be reported in subsequent papers and at that time more data will be given about the other methods of procedure with other rats.

These females were kept in individual cages and a daily vaginal smear was taken. At the same time the food put in and the food taken out uneaten from the previous day was weighed. In this way daily food consumption was accurately known, and the actual amount of any given constituent could easily be computed. The rats themselves were weighed each day. When the female was found to be in Stage 1, 2 or early 3 a fertile male (on stock diet) was introduced. If spermatozoa were found next day, he was removed—if not, he was left until Stage 5 smears were found, and then removed. This was repeated until pregnancy took place. The occurrence of pregnancy was checked by continued Stage 5 smears, placenta sign, gain in weight of mother, and palpation. When young were born they were individually weighed on the day of birth, but thereafter only the total litter weight was recorded each day. At first the whole litter was allowed to nurse, but with the reduced diets this was found to be too much of a strain on the mother, with subsequent failure of the young to develop to weaning. Therefore after the second month the litters were all cut down to six at birth.

Following Simmonds' work, and also using a diet similar to that in calcium experiments by the author[4] it was determined to use 20 per cent protein in the control diet (I). This was before Slonaker reported optimum performance on 14 per cent though, as noted above, there is some doubt as to whether the adverse effects reported by him are actually the result of the increase in protein or of the decrease in vitamin content. Simmonds' diets very high in protein permitted normal reproduction and lactation.

The actual record was as follows:

On Diet I (protein 20.8 per cent with lard as source of fat) nine out of nine rats (one died under ether) promptly became pregnant, but there were four absorptions (due to the fact that the lard destroyed the vitamin E). Five litters of young were born with 44 young. Only one mother was able to nurse her young and she raised nine out of eleven with an average weight of 27.7 Gm. at 21 days. Because of the fact that this diet was deficient the results may be eliminated from this part of the discussion. Diet I unless specifically qualified will be understood to have the analysis recorded in Table I of Protein 20.8 per cent and Butter 16 per cent.

TABLE I

ANALYSES OF DIETS

	Diet I	Diet II	Diet III	Diet IV
Casein	20	16	9	4
Salt Mixture	4	4	4	4
Starch	51	55	57	62
Wheat germ	5	5	10	10
Yeast	2	2	2	2
Cod liver oil	2	2	2	2
Butter	16	16	16	16
	100	100	100	100
	Protein 20.8%	Protein 16.8%	Protein 10%	Protein 5%

Corrected Diet I (protein 20.8 per cent—butter 16 per cent). Ten out of ten mothers became pregnant giving birth to 102 young. Only five mothers were able to nurse their young, raising 33 out of 47 to weaning. The average weight of the young was 36.6 at 21 days.

This did not seem to be a very good result on what was supposed to be a normal diet. An attempt was therefore made to improve Diet I still further by adding a small amount of lettuce each day (about 10 Gm.). With this change these same rats again became pregnant giving birth to 109 young. This time eight of the ten were able to nurse their young, raising 58 out of 73 to weaning. The average weight of the young at weaning was 36.2. While this record of performance was not perfect, part of the

*These changes were instituted because of the fact that the original diet was low in vitamine E and consequently a large number of absorptions took place. Lard was used as the first fat in the proportion of 20 per cent. This neutralized the E as reported by Evans[3]. At the time we were not aware of this effect of a rancid fat and tried increasing the wheat germ and adding lettuce as well as changing the lard to butter. See Part III for details.

trouble seemed to be due to the fact that, because of some individual peculiarity, two mothers were never able to raise a litter no matter how many pregnancies occurred or what the diet was. On the same diet another group of ten mothers all became pregnant, giving birth to 83 young. The litters were cut down to six (whenever the number at birth exceeded that figure) and 52 young out of a possible 57 were raised to weaning. The average weight of the young at weaning was 42.2 each. (This was in part due to the cutting down of the number of nurslings per mother to an average of 5.7 young each.)

On Diet II (protein 16.8 per cent, with butter) nine out of nine became pregnant, giving birth to 86 young. Only six mothers were able to nurse their young, raising 46 out of 47 with an average weight at weaning of 31.5 Gm. each, an excellent performance.

On Diet III (protein 10 per cent with butter) ten out of ten became pregnant, giving birth to 74 young. Again only six mothers were able to nurse and raised 37 out of 45 to weaning with an average weight for the young at weaning of 30.1 Gm. each. The performance here also was surprisingly good.

On Diet IV (protein 5 per cent with butter and lettuce) nine out of nine became pregnant, giving birth to 70 young. On this diet only five mothers nursed, raising 22 out of 28 to an average weight of 19.8 in 21 days.

When put back on *Diet I* (protein 20.8 per cent with butter and lettuce) seven out of seven became pregnant, giving birth to 49 young. Six mothers were able to nurse, raising 29 out of 30 in 16.6 days to an average weight of 39.8 Gm. each! Such a performance speaks for itself.

Turning now to the amounts of food eaten and the calculated analyses we obtain some very interesting figures. These are shown in Table II. On all diets the amount of food eaten was about the same during pregnancy and contained roughly the same number of calories. The amount of protein, however, decreased progressively from a total daily amount of 2.90 Gm. to 2.52, 1.44 and 0.83. On these amounts the litter size decreased also from approximately 11 through 9.5 to an average of 7.5±. Even returning these animals to a normal protein did not bring the litter size back to the original figures. The average weight of the individual young at birth has been found to vary with the litter size. The reduction in the amount of protein in the mother's diet did not change this until the minimum protein intake of 0.83 was reached; on this diet the young were not quite up to standard, but the difference was trifling. Furthermore, the mothers' usual gain during pregnancy was practically the same on all diets. To summarize: reduction in the amount of protein had little apparent effect upon conception and pregnancy

TABLE II

SUMMARY OF DATA

		Pregnancy						*Lactation*							
		No. mothers	Daily amt. food	Daily protein	Daily calories	Mothers' total gain or loss for 21 days	Litter size	Average weight	No. mothers	% mothers able to nurse	Daily amt. food	Daily protein	Daily calories	Mothers' total gain or loss for 21 days	No. nursed per mother at start	% raised	Ave. weight young at weaning
Diet I.	Protein 20.8	10	14.5	2.90	68	+21.3	10.9	5.25	8	80	24.2	4.84	114	—4.0	7.62	93	36.2
Diet II.	Protein 16.8	9	14.8	2.52	69	+28.3	9.5	5.25	6	66	25.4	4.32	119	—19.3	9.0	85	31.5
Diet III.	Protein 10.	10	14.4	1.44	66	+29.5	7.4	5.57	6	60	21.6	2.16	99	—40.5	7.0	88	30.1
Diet IV.	Protein 5.	9	14.9	0.83	69	+22.9	7.7	4.98	5	55	15.4	0.86	72	—74.0	5.6	78	19.8
Diet V.	Protein 20.8	7	14.1	2.82	66	+27.5	7.0	5.10	6	87	19.0	3.80	89	+6.3	5.0	96	39.8

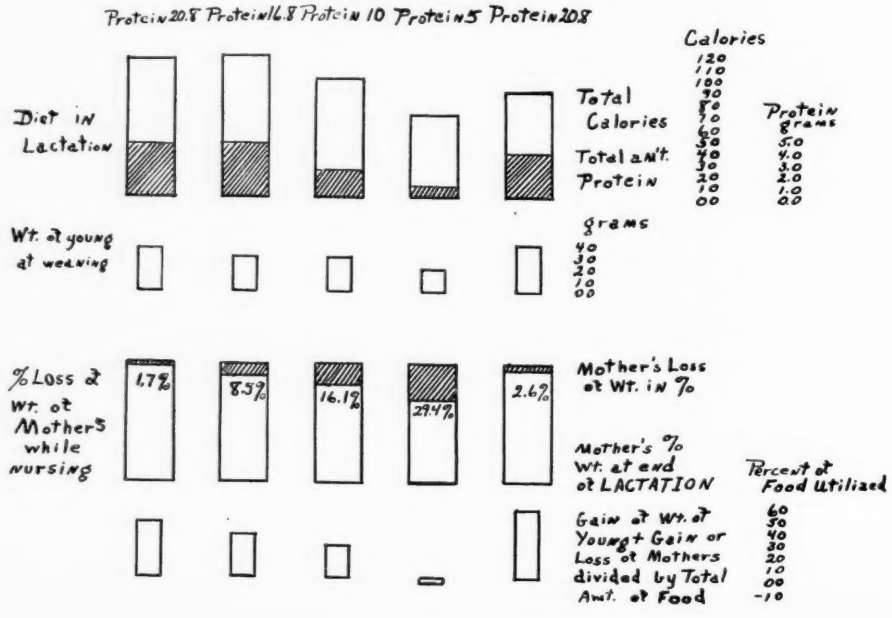
beyond a reduction in fertility, and a slight effect upon the weight of the young when the reduction was extreme.

The effect upon lactation was much more striking and in this respect duplicates the result of the similar work previously done on calcium[4]. Though the reduced amount of protein had shown so little apparent effect on pregnancy, something had been taking place as is well shown by the decreasing percentage of mothers able to nurse at all. This fell from 80 per cent progressively to 55 on Diet IV and rose to normal, or even above, when these animals were replaced on Diet I. The amount of food eaten (in grammes) was roughly three times the number of young nursed except that on Diet IV it was decidedly less than this and, as if to compensate, was a great deal more when the animals were put back on Diet I. The calories followed the amounts eaten. The total daily protein dropped from 4.84 through 4.32 and 2.16 to 0.86. The mothers lost a progressively greater and greater amount of weight until returned to Diet I. Altogether there was a much better performance than could have been anticipated. On Diet I both before and afterwards 95 per cent of the young which began to nurse were raised to weaning. On Diets II and III the average was still high—85 per cent for 9 and 88 per cent for 7 respectively. On Diet IV in spite of a reduction to 5.6 nurslings each, only 78 per cent were raised to weaning. Even more striking was the effect upon the weight at weaning (21 days). This fell from the normal of 35 or better to 30 for Diets II and III and 20 for Diet IV.

In studying these results it is evident that in spite of very drastic changes in the protein, the fetus or the nursling suffers relatively little, and that it is the maternal organism which, by drawing on its reserves, is able to make up for the lack in the diet. Compare the loss for the mother during the three weeks of nursing with the average weight of the nursling on Diets I, II and III. On Diet II the loss is five times as great, and on Diet III ten times as great, while the young weigh only about 16 per cent less! On Diet IV the amount of protein ingested is so slight that even a loss eighteen times the normal is unable to protect the young and they weigh 44 to 50 per cent under what they should.

Some of these changes have been shown graphically in Chart I. This needs no comment beyond that already given, except that the last row of graphs has been presented to show in a rough way the relationship between the amount of food eaten and the results obtained in maternal and filial loss or gain. To denote these changes the term metabolism has been used in its most general sense, which relates more to material transformations than to any energy changes which took place. These latter were not measured and no estimate of them can be given. It seemed, however, worth while making some simple calculations in regard to the former changes. No account was taken of water, but daily gain of mothers plus fetuses or nursling was figured as the numerator of a fraction of which the daily amount of food eaten was the denominator. The fraction expressed in percentage form is called the "metabolism". Two facts stand out, first, that the metabolism (per cent of food utilized in growth) is much

CHART I



higher in lactation than in pregnancy and, secondly, that the metabolism of lactation is much more adversely affected by a lessened protein intake than is that of pregnancy.

SUMMARY. On a diet containing 20 per cent protein, where the daily consumption was 2.9 Gm. of protein, fertility was optimum. Progressive reductions in the daily protein intake to a minimum of 0.83 Gm. reduced fertility. These changes did not, however, materially affect pregnancy. This failure to affect pregnancy in the rat should not, however, be interpreted as meaning that pregnancy might not be affected in a larger mammal where the young are born at a later stage of development.

The effect upon lactation was very definite. As the daily amount of protein ingested fell from 4.84 to a minimum of 0.86 Gm. a smaller percentage of young was raised to weaning, the average weight of the young at weaning was less and the loss of weight suffered by the mothers during lactation was greater. Finally, with the reduction in the amount of protein eaten the "metabolism" during lactation fell below zero.

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THE BOSTON LYING-IN HOSPITAL FIFTY YEARS AGO

BY ALFRED WORCESTER, M.D.*

MY interneship in the Boston Lying-In Hospital began late in March, 1883. I had applied for the service beginning in July; but as my predecessors had resigned, because of family disasters, I had to take it then or never. The term of service was supposed to be two months as junior, and then two as senior house officer, as internes then were called. But my juniorship lasted less than a fortnight. The following month I was in sole charge. Then I was demoted, in order that a classmate might have the senior service for the next six weeks. He had even less experience than I, but he was too good-natured to interfere much with my management. In July, I was again the senior, and this time with a first-rate classmate as junior.

Even after a half-century I am in some doubt whether I ought to tell the frightful story of the pestilence that caused the temporary closure of the hospital. Perhaps the bare records, in my own handwriting, are sufficient. But those musty record-books are available only to antiquarians; and, even if published and read between the lines, without direct testimony how can younger obstetricians realize the horror of the septic era or appreciate the blessings of modern aseptic methods? Moreover if, as I suppose, I am the sole survivor of that period I am now the only one who can tell the story. Such are the considerations that lead me to attempt it.

The hospital at 24 McLean Street was ten years old, or rather it was then ten years since its revival. An adjoining and similar dwelling house had lately been added, and the intervening walls pierced for doorways. On the first floor, partitions having been removed, were two wards of six beds each. On the second floor the old chambers held two beds, or three on a pinch. About twenty patients could thus be accommodated, and a few more could be crowded in. In the attics were rooms for half as many nurses and for the two house officers. The matron, with her husband and small son lived in the basement. There, too, were the kitchen and nurses' dining room. The house officers had to take turns at some neighboring boarding house for their meals.

Before attempting to tell what went on in the hospital I must first describe the personnel. The trustees I never saw. Their only function, so far as I understood, was to raise the necessary funds for carrying on the hospital and to back up Dr. W. L. Richardson in his management of it. Dr. W. E. Boardman was his colleague. They had alternate terms of three months each as visiting physicians. I shall later bring them into my story. Dr. C. M. Green was then the assisting visiting physician. There were, besides, two other assistants who might be called in. Only one of them, Dr. R. B. Dixon, did I ever see, and I saw him only once.

The matron, Mrs. E. J. A. Higgins, was a wonderful woman, an efficient housekeeper, an excellent midwife, and a great-hearted loyal friend. Her sympathy often made her teary, but never blinded her judgment. She had such perfect self-command that her supremacy was unquestioned. Even when an explosion might well have been forgiven, she could hold her tongue and bide her time. Since the rebirth of the hospital she had been its mainstay. She knew all about it and all about those who had ever had anything to do with it. Her memory of past patients was marvelous and her kindness

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to them boundless. Because in those days the patients were almost always illegitimate mothers, they were in dire need of friendship and subsequent help. There were two Boston women, Miss Putnam and Miss Parkman, who would always answer calls for this help. The hospital had no other friendly visitors. Even the flower mission passed by on the other side.

The nurses were a queer lot. Against the headnurse's temper I had been warned by one of my predecessors. She had once slapped his face with the wet washcloth at a patient's bedside. This he told me without explanation of his acquiescence, although it was then quite within the house officer's province to discharge a nurse for insubordination or misconduct. None of them were graduate nurses. Some had been dismissed from the training schools in other hospitals. One of them had been a failure as a school teacher. Another was beginning her career as an adventuress. But others were honest hardworking women who after a six-months' course would be given certificates as "monthly nurses".

My own unfitness for the responsibility thrust upon me can be at least partially explained by the deficiency of my medical education. I had then had the only eleven months that I ever had in the Harvard Medical School. This was the way of it: The first year of the course I had escaped by a few weeks' cramming for examinations in anatomy and physiology. My college chemistry sufficed. The last half of the second year I had lost, and also nearly my life, because of "dissecting room poisoning". And now, after six months of my third year, and so after only two-thirds of the lectures in obstetrics, I was turned loose in the Lying-In. I had had charge of three outside obstetric cases, one of which was a forceps delivery by another medical student. And I had seen a few normal deliveries in the hospital by my predecessor.

In several previous years the hospital had had to be closed because of prevalent septicemia. Dr. Boardman's term of service was just ending. In a few days Dr. Richardson's term was to begin. As both were actively engaged in private obstetrical practice, naturally neither relished service in a hotbed of what then was called childbed fever. The reluctance of the visiting obstetricians to serve at these times was thus a dim reflection of what Oliver Wendell Holmes and Semmelweis had taught. But in Boston there was as yet only slight recognition of Pasteur's discoveries. The only reference to the germ origin of disease, which I had heard in the Medical School, was "Let those who will believe in it; I do not."

Yet some of the Boston surgeons, as partial followers of Lister, were soaking their reef-sponges in carbolic solutions and clouding their operating rooms with carbolic spray. But they still wore their blood bespattered Prince Alberts. Carbolic acid solutions were also used in the Lying-In, but only for intra-uterine douches in cases of puerperal fever, or as general deodorants. The stench not only permeated the hospital but also became a cause of complaint in the neighborhood.

During the few days of my juniorship I was given more or less complete care of twenty odd patients who had been delivered by my predecessor. At least half of them were septic, but only one died. During the next twenty five days I delivered twenty-five women, seventeen of whom became seriously septic. Five of them died, one on the third day, one on the fifth, and the others nearer a fortnight after delivery.

The virulence of the infection seemed to be steadily increasing. Generally the first sign of it would be a terrific chill with high fever immediately following. In some cases the fever would be continuous, as it is in cases of lobar pneumonia. In others there would be repeated rigors, with rectal temperatures varying many degrees within even an hour. In one patient this variation was once from 93° to 105°, and nearly as much several times afterwards. To our surprise she recovered. Some of the patients became maniacal; with one I had a furious fight to prevent her escape from the hospital. Only a few weeks previously a patient had leaped from her bed, dashed through the window and raced up the street. In some cases there was a profuse diarrhea, and to this contagion neither nurses nor doctors were immune.

Peritonitis was almost, but not always, evident in the fatal cases. Some sank to their death with far less suffering than fell to the lot of those who survived. That so many of the patients became septic and that so many of them died is not so much of a wonder as it is that any escaped. Against those poor women the dice were loaded, for besides the ordinary perils of childbirth, deadly dangers awaited them in that charnel house. Such conditions as existed then are now in these aseptic days hardly imaginable. And so, from the films of my memory, let me picture for you a typical case.

When K. F. applied for admission, as she was not in labor, she was referred to one of the miserable boarding houses near by, there to wait until her pains began. Prenatal care had not been heard of then. She had no preliminary examination, not even a urinalysis. When brought to the hospital she had to pass, in the narrow hallway, the coffin of one of her predecessors waiting there for pauper burial. The hospital had no other morgue or other entrance. Ominous that, of the invisible and yet terrible dangers she must soon encounter! As there was no delivery room she must be put to bed alongside women already deathly septic. Still worse for her, she must at once undergo vaginal

examination by hands from which no washing could remove the stench of the intra-uterine douches lately given. As her labor progressed such digital examinations would again and again be made. Anesthetics would not be given her until after delivery when her torn perineum would be repaired with cruel silver sutures, which would torture even more when her turn came for intra-uterines. If her labor pains slackened she probably had large doses of quinine, which again would be given her and with even less effect when the septic chills began a day or two later. Drugs in those days were given freely; ergot almost always, morphine for after-pains, bromide and chloral for sleeplessness or delirium. The only real relief that I remember was given in this case was the double Y breast bandage which had been invented by my predecessors*.

Who then could say how this poor girl received the massive infection from which she died on the fifth day? Was it from the sickening odors of the ward, as was then supposed? Alas, we now know that not one of the hospital utensils, not an instrument, and above all neither her nurses nor her doctor ought to have been allowed near her defenceless bed.

This tragic story would be incomplete without telling why she was only too willing to die. She was the wife of a British captain. In his long absence she had yielded to the seduction of a former lover. Then she fled to this country. Her jewelry had been sold, her wedding ring was in pawn; had she survived, Tewksbury would have been her only refuge, and it was at this time that the gruesome descriptions of that almshouse were being published by Governor Butler. In fact, he was then trying to find out what was done with the dead babies from the Lying-In, and his agents were threatening me for my defiance of his requisitions. Even now I am not sure what became of those tiny corpses which were delivered to undertakers supposedly for pauper burial. Our more serious misgivings concerned the well babies after they left the hospital. Not seldom the police would bring back a dead baby vainly expecting from us the incrimination of its mother.

When once I had been charged by the visiting physician with having caused a baby's fatal rectal hemorrhage, I carried the body to the morgue of the Massachusetts General Hospital where the kind pathologist freed me from blame. And once again I took advantage of his kindness, in order that I might be shown the pathology of childbed fever. At dead of night we carried the corpse through the deserted streets. At the gates it seemed as if we could never wake the janitor without waking everybody else, and when once inside, before we reached the morgue, several times from sheer fatigue we had to lay down the stretcher. I shall never forget that autopsy—the belly full of pus, with multiple abscesses everywhere.

This patient died on May third. Others died on the ninth, eleventh, twelfth and fourteenth of the same month. As many more were on the danger list. Only one of the patients in the hospital at that time could be considered free of septic fever and her temperature was only just under 100°. Evidently it was high time for radical measures. But instead of closing the doors to new patients, those already in the hospital were moved about so that the first-floor wards in turn could be repainted and refurnished. It was at this time that I was demoted. As I look back I do not wonder that another was given the care of incoming patients. There was thus the chance of staying the pestilence. But the implication of my accountability for it was not easy to bear. That, however, was of small consequence in comparison with the hopelessness of the situation. For the newly furnished wards were soon filled with septic patients. The most that could be said was that their infections seemed less severe. In fact, so far as my notes reveal, all of the next sixteen patients, who were delivered by my associate, left the hospital alive. But the continuance of the infection, even in the refurnished wards, made the closure of the hospital imperative. Dr. Richardson's previous record was broken. It was no small relief to me, as his scapegoat, when he sailed for Europe. I doubt if he ever knew how much I had been hurt. At any rate, in subsequent years I found him as kind and helpful as he was then the opposite. It is no wonder that he was disgusted and discouraged. For when the hospital was reopened, after most thorough and expensive renewal of all that was in it, the second patient admitted soon became septic. We were all in despair.

On one of those dark days a former house officer, Dr. Rufus Kingman, called. He had just returned from Prague where, as he told us, puerperal fever had been eliminated by thorough disinfection of the obstetricians. "With what?" we asked. "Bichloride of mercury, one to a thousand of water," he replied. This was immediately ordered, but Canning and Patch soon answered that the poisonous drug would not dissolve in water. When told that it did in Prague and must do so in Boston, they finally reported that it would dissolve if soda were added. And soon they began sending to the hospital a daily supply of the solution.

Not many days afterwards Dr. Richardson on his rounds asked, "What's in that basin and what's that nail brush for?" "That, sir, is a solution of corrosive sublimate to scrub hands with before making a vaginal examination."

Although I well remember his words, it will

*Drs. Kingman, Otis and Hayward. Nothing else that I learned in the Lying-In has been of such continuous value in my subsequent practice. If the proper application of this bandage, once the pride of the Lying-In nurses, has become obsolete, it ought to be recovered.

be enough if I quote the gist of his remarks. I was told to throw away the solution, lest as my next folly I should be scrubbing perineums with it. He certainly had the gift of prophecy.

Another order of Dr. Richardson's which I disobeyed was that I should stop giving class lessons to the nurses. I could not obey that order. The facts were these: Formerly the Lying-In Hospital nurses were supposed to learn by carrying out verbal orders, without any explanation of the reasons; and in their work they were to be commended or criticized by the house officers. Of course, in such a régime, demoralization was inevitable. Having known something about the reformation of nursing in American hospitals during the previous ten years, I determined to copy the methods of the new training schools. Mrs. Higgins was very willing to help. And I told the nurses that hereafter my comments upon their work would be given to them only through the matron; but also that in class lectures I should try to teach them the principles of nursing. Had I thrown a bomb into their quarters, no greater explosion would have followed. They immediately signed the request that, inasmuch as I would no longer be willing to discuss matters with them in the wards, all of our future communications should be in writing. I of course agreed and at once began to write my orders, the first of which was that the nurses should keep clinical records. This, as I believe, was the beginning of what soon became the customary duty of nurses. My second order was for them to meet for a weekly lecture. And this was the beginning of class instruction in the Lying-In Hospital. Afterwards I published my lectures, the earliest of the subsequent flood of books on obstetrical nursing.

When the renovated hospital was opened, June 26, 1883, I was again in sole charge. The first patient made a normal convalescence. The second, as I have already said, became unmistakably septic, but recovered. And that is the way it went on for the next two months. Of the thirty odd patients admitted about every other one had at least several days of fever. But the virulence of the infections was so much less than it had been that we all felt easy over conditions which nowadays would occasion grave concern.

As I have no record of when we began scrubbing our hands in the bichloride solution, it is not possible to decide how much that procedure had to do with the lessening of the infections. There were plenty of other ways than by the doctor's hands, by which infections could be carried. And when I read in my notes how often the repair of the torn perineum was only partially successful, and when I remember how many years afterward it was before our instruments were disinfected, my only wonder is that any of that stitching held. But, besides hand disinfection and carbolic douches, we were using, that summer, iodoform for dusting all lacerations and also for intra-uterine insufflations whenever symptoms of sepsis appeared.

Dr. Boardman, the visiting physician, was spending his summer at Hull. He called every day at noon. Occasionally he walked through the wards, but he never interfered. Otherwise I was having my own way: Perhaps I was operating too often. But at the time I was fully persuaded that these operations were for emergencies. And there was the further excuse that the visiting obstetricians were too far away. Telephones in those days were very temperamental; when most wanted they were seldom available, and, in fact, the only sure way of getting a doctor was to send a livery stable carriage after him.

As I now read my notes of versions, forceps deliveries, adherent placentas, postpartum hemorrhages, etc., I recognize how defenceless I was against the charge, had it been made, of meddlesome midwifery.

Besides the sixty-odd patients for whose deliveries I was responsible, during my term of service I had more or less charge of forty others. The maternal mortality I have already stated; that of the babies I have never wanted to compute. But this much can be said, that, although it was not until after I left the Lying-In that I ever saw a mother rejoice over the birth of her child, we nevertheless tried to save the babies and to persuade their mothers to take care of them. That, be it remembered, was fifty years ago. And not half so long since, in the maternity wards of a great city hospital, I found that the illegitimate babies were never given breast milk but, instead, such a starvation formula of watered cow's milk that no more than one in ten left the hospital alive. Even that was better than feeding them on powdered glass, as was done in a so-called home for the unwanted babies of another city.

In comparison with present hospital conditions those of a half century ago were awful. And yet, in comparison with other accommodations then available for one whose illegitimate pregnancy made her an outcast, the Boston Lying-In was a haven of mercy. She would find there unstinted kindness and at least all the professional help that we knew how to give. For our ignorance only were we to blame.

MEDICAL PROGRESS

REVIEW OF GASTRO-INTESTINAL SURGERY IN 1932

BY M. A. MC IVER, M.D.*

THE following is a brief survey of some of the more important contributions to gastro-intestinal surgery during 1932.

STOMACH AND DUODENUM

Peptic Ulcer. The etiology of peptic ulcer has long been a baffling problem to both pathologists and clinicians. In recent years the idea that either an infection, or disturbance in the acid-base equilibrium of the upper intestinal tract, is responsible for these lesions has held a predominant place in the literature. During the past year, Cushing[1], his interest in the subject aroused by the occurrence of acute, perforative lesions of the stomach and duodenum early after certain of his operations for cerebellar tumor, has revived the older, neurogenic theory as to the etiology of peptic ulcers. This theory, Cushing points out, was advanced by Rokitansky as early as 1841, but was later overshadowed by the views presented by Virchow, who championed the idea that ulceration was essentially a local process.

Cushing has been able to assemble from his clinic 11 cases (to which he has added others, collected from the literature) that have a bearing on the neurogenic theory of ulcers and erosions of the upper intestinal tract. The clinical evidence may be summarized as follows: first, lesions have been seen to occur as a sequel to certain intracranial operations; secondly, they occasionally accompany intracranial tumors; and, thirdly, ulcerative processes occur in the newborn in association with cerebral birth injuries or intracranial disorders of other kinds. In addition to an analysis of the clinical findings, Cushing's article carries an account of the experimental work on ulcers produced by nervous stimulation or degeneration, and a comprehensive description of the physiology of the sympathetic and parasympathetic nervous systems as related to the etiology of ulcer. The author points out that "Those favorably disposed toward the neurogenic conception of ulcer have in process of time gradually shifted the burden of responsibility from the peripheral vagus to its center in the medulla, to the midbrain, and now to the interbrain, newly recognized as a highly important long overlooked station of vegetative impulses easily affected by psychic influences."

Those who are interested in the neurogenic theory of ulcers should read also Fulton's[2] article, which presents a further discussion of the physiology of the hypothalamus; and a paper by Best and Orator[3], who conclude on the basis of their experimental work that direct, continued stimulation of the vagus with a known irritant does not produce acute or chronic gastric or duodenal lesions, and that consequently section of the vagus nerves for the treatment of peptic ulcer will prove of little value.

The etiology of gastric and duodenal ulcers is also discussed by Matthews and Dragstedt[4], who consider that the chemical action of the gastric secretion seems to be the most important factor in the production of an acute lesion and in its subsequent chronicity. The etiology of postoperative peptic ulcers is dealt with in a paper by Steinberg and Proffitt[5]. These authors believe that infection plays only a secondary rôle in the formation of these ulcers. They advise a large anastomotic opening between the jejunum and fundus of the stomach as the best method of preventing jejunal ulcerations.

Only those who have delved into the literature of the etiology of peptic ulceration can realize how much confusion exists. One of the disconcerting features is the apparently unlimited success of practically all investigators in producing experimental peptic ulcers, whatever procedure they have employed; most of these experimental ulcers, however, have been acute, and thus not really comparable with the chronic peptic ulcer encountered in human beings.

Acute perforations of peptic ulcer are analyzed by Converse[6] in a study based on 106 cases. Converse recommends conservative treatment by simple closure of the perforation; his operative mortality was 29 per cent. Recurrent perforation is discussed by Pearse[7], who has reviewed 4,183 cases and found 33 instances in which more than one perforation occurred. This author reports the interesting finding that of the 24 cases of perforation treated by suture alone, only one had more than two acute perforations; whereas of the eight cases treated by primary gastroenterostomy, five had three or more perforations, a jejunal ulcer being responsible for the perforation in three of these cases.

The prognosis of gastric and duodenal ulcer is discussed by Moynihan[8] on the basis of his tremendous experience. He emphasizes the fact (which is often overlooked) that treatment should not stop with the operation; and his

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article carries recommendations as to general rules of hygiene and diet to be followed for at least one year after operation.

The treatment of advanced gastrojejunal ulcer is discussed by Estes[9] and a description of his operative procedures in this serious type of lesion is presented. In one case the operation consisted in detaching and closing the stomach and jejunum, leaving the ulcer still attached to its mesentery; the stoma was cauterized; the result was satisfactory. In the other case reported, after detaching the stomach from the upper border of the stoma and closing it, an anastomosis between jejunum and jejunum was carried out side tracking the intestinal stream around the ulcer area, with a satisfactory result.

The treatment of gastric and duodenal ulcer is reviewed by Miller[10] in an article carrying descriptive illustrations of the various types of operation that may be employed. The author stresses the fact that in dealing with gastric ulcers one must be suspicious of malignancy. Duodenal ulcers he advises treating by medical measures until the patient elects surgery, and then doing the simplest type of operation.

The choice of surgical procedure for duodenal ulcer is also discussed by Walters[11]. European surgeons, as is well known, have in general performed a more radical operation for duodenal ulcer (involving the resection of a considerable portion of the stomach) than have the surgeons in America. Walters brings up the point that this difference in treatment may be based on the difference in the pathological lesions encountered: in Europe, gastritis is more commonly associated with duodenal ulcer than it is in this country, and therefore the more radical operation is perhaps more justified.

That duodenal ulcers having recurrent hemorrhages present an extremely serious problem, is well known. Balfour[12] discusses operation upon these cases and points out that it is impossible to attempt to establish any routine method of treatment: the type of operation best suited to the individual case should be selected.

Among the articles dealing with technical procedures for dealing with peptic ulcers, a paper by Rienhoff[13] on infrapapillary gastroduodenectomy by mobilization with retromesenteric displacement of the duodenum and jejunum, deserves special mention. In this operation, in carrying out the anastomosis between the stomach and duodenum, instead of using the second portion of the duodenum, as is customary, the third, or horizontal, portion is selected. Among various advantages claimed for this method is the fact that the horizontal portion of the duodenum is not subject to ulcers and therefore is not likely to be found bound down by adhesions, and also that this procedure is considered to afford the maximum drainage of the stomach and to promote a more efficient reflux of bile, pancreatic secretion and duodenal secretion into the stomach. The article carries complete illustrations of the technique, and reviews the history of gastroduodenostomy.

Carcinoma of the Stomach. An analysis of 417 patients with carcinoma of the stomach admitted to the Presbyterian Hospital, Chicago, is presented by Gatewood[14]. Fifty per cent of the patients were inoperable at the time of admission. Among the remainder, the mortality following gastroenterostomy was 26 per cent; following gastric resection 32.6 per cent. Of the 58 patients on whom resection was carried out, five were living eight or more years after the operation.

Benign Tumors of the Stomach. Benign tumors of the stomach are discussed by LaRoque and Shiflett[15], who point out that although this is a relatively rare condition, approximately 1,000 such cases have been reported in recent years. LaRoque's article carries a discussion of the points in diagnosis of this lesion, which is essentially based on x-ray examination.

Duodenum. Congenital hypertrophic pyloric stenosis in infants is the title of a paper by Donovan[16] based on the study of 104 cases. The author stresses the importance of preoperative treatment, chiefly measures combating dehydration, and the use of blood transfusion where indicated, and his figures show a great lowering of mortality since this has been perfected.

Ten cases of congenital obstruction of the duodenum in children are reported by Ladd[17]. He divides these obstructions into two groups: first, the intrinsic or intramural type, caused by septa within the lumen of the bowel; and, secondly, the extrinsic or extramural type, caused by pressure on the outside of the bowel, and dependent, Ladd considers, on an incomplete rotation of the bowels. The author points out that while vomiting is an important feature, it may be extremely intermittent in character, the patient going several weeks or even months without exhibiting this symptom. The operations recorded are posterior gastrojejunostomy, posterior duodenojejunostomy, reduction of midgut volvulus, and freeing of cecal attachments, depending upon the type of obstruction found. Related to the above lesions are congenital duodenal adhesions, which are discussed by Knox[18]. The author considers that this condition should be differentiated from pylorospasm, pyloric stenosis, and atresia of the duodenum. Operative treatment is advised.

Two unusual lesions of the duodenum are discussed by Matthews, Delaney and Dragstedt[19] and Bookman[20]. Matthews, et al., report a case of tuberculosis of the duodenum, and review the literature; Bookman discusses carcinoma in the duodenum arising from aberrant cells from the pancreas; he also reviews the literature brief-

ly. Chronic obstruction of the duodenum caused by enlarged peritoneal glands is the subject of a paper by Penick[21]; his article carries illustrations of the x-ray picture found in this type of pathology.

INTESTINES

Acute Intestinal Obstruction. During the past year a number of statistical articles dealing with the subject of acute intestinal obstruction have appeared. Among these may be mentioned those by Cornell[22], who reported 235 cases with a mortality of 51.48 per cent; by Koslin[23], who reported 185 cases, mortality 38.8 per cent; by McIver[24], reporting 335 cases, 31 per cent mortality; by Morton[25], 105 cases, 28.5 per cent mortality; by Vick[26], 6,892 cases, 26.2 per cent mortality; and by Vidgoff[27], 266 cases and 45.9 per cent mortality. In looking over this large group of statistics, several factors stand out. First, the pathological lesions discussed under the heading of acute intestinal obstruction are varied and there is no uniform method of grouping the cases. Secondly, due to this fact, the percentage of mortality reported can be varied within wide range depending upon whether certain groups of cases, for instance, obstruction by strangulated external hernia, or obstructions where peritonitis complicates the picture are included or excluded. Thirdly, bearing this in mind, it is very difficult to show that the mortality from this disease is any lower at present than it was in the previous decade. The series from the Massachusetts General Hospital showed no lowering when the present decade was contrasted with the preceding one; and Cornell, reporting cases from the New York Hospital, where more or less the same method of grouping cases has been maintained for a number of years, makes the statement that his statistics agree closely with statistics compiled 30 years ago.

Evisceration and surgical evacuation of the small intestine in certain desperate cases of obstruction are advised by Cheever[28], who does not feel that this procedure is as likely to produce surgical shock as has been generally believed. He reviews the literature dealing with this subject.

Obstructions in acute peritonitis are discussed by Robb[29], who considers that most of these are functional and have no organic basis, and that areas both of spasm and of atony are present. His article carries an account of his clinical management of these patients. Obstruction caused by food is discussed by Elliot[30]; he reviews the literature and adds a case report. Three cases of internal hernia are reported by Steinke[31].

The subject of enterostomy is reviewed by Shelley[32], who presents an extensive résumé of the literature. He is not able to arrive at any definite conclusion as to the effect of this procedure on the mortality rate; but this is not surprising considering the widely different conditions for which the operation is performed, and especially considering the fact that it is often used as a forlorn hope for a desperately ill patient.

A case in which feeding was carried out with gratifying results by placing nutrient fluids in a container and allowing them to flow by gravity into the intestine, utilizing a jejunostomy opening, is reported by Stewart[33].

Diagnosis of acute intestinal obstruction by x-ray without contrast media, has received an increasing amount of attention in the recent literature. It was mentioned in several of the articles noted above, and has been the special subject of articles by Swenson and Hibbard[34] and Ginzburg[35]. Although the method has its limitations, it would seem to be of definite benefit in certain cases. The articles by Swenson and Hibbard and Ginzburg carry illustrations, and suggestions as to the technique to be used. It is, of course, not necessary for the reviewer to stress the danger of giving barium by mouth where there is any suspicion of intestinal obstruction.

The x-ray diagnosis of subacute and chronic intestinal intussusception is discussed by Sussman[36] in a very illuminating article which carries illustrations of the x-ray appearance of these various lesions. It should be mentioned in this connection that several other articles dealing with intussusception have appeared, among them papers by Robbins[37], Peterson and Carter[38] and Shelley[39]. The latter authors stress the use of the barium enema as an aid to diagnosis. Shelley considers that where the intussusception cannot be felt by ordinary methods it may frequently be made out if the child is held in the sitting position while a rectal bimanual examination is carried out. All these authors discuss the question of the best method of dealing with irreducible intussusceptions where resection is not feasible. The anastomosis between the terminal ileum and transverse colon would seem to have a place in these difficult cases. Robbins reports an instance of the successful application of Brown's method, where an incision is made into the constricted ring of the intussusception; it seems to the reviewer that the drawback to this method is the great danger of peritonitis.

Intussusception in adults is discussed by Moore[40], who stresses the point made by previous writers that some type of tumor or Meckel's diverticulum is frequently an etiological factor in these cases.

Small Intestine. Several articles dealing with the subject of tumors of the small intestine have appeared, among which should be mentioned two by Raiford[41] and Cave[42]. Tumors of the small

intestine are of course rare: Cave reports 2 cases; Raiford, after a careful study of the literature, was able to collect 339. Raiford presents pictures of numerous specimens of the tumors encountered, showing their gross and microscopic pathology, and gives a diagram of their location; the article carries an extensive bibliography, and should prove extremely useful for reference.

Lymphosarcoma of the alimentary tract is the subject of articles by Ullman and Abeshouse[43] and Cheever[44]. Cheever urges exploratory operation and biopsy, in order that x-ray therapy may be carried out upon the cases that prove to be lymphosarcoma, and in order to obtain material so that the histological picture may be correlated with the results of the radiation. Ullman and Abeshouse advise radical resection of the involved gut and mesentery where this is possible. They consider that radiation should be employed in every case: in the inoperable cases to control the growth and prolong life; in the operable to prevent recurrences and metastases.

The pathology and treatment of intestinal fistulae have been discussed by several authors, notably Potter[45] and Baker[46]. Erosion of the skin by the digestive juices, which is frequently a serious problem, is particularly troublesome when the fistula is high in the intestinal canal. Potter has found it useful to keep the patient in a prone position on a Bradford frame, so that the secretions from the fistula may drop directly into a container below and do not form a puddle on the skin. (Constant suction applied to these fistulae is frequently a useful procedure, and often accomplishes the same result.) Baker's article deals particularly with the various types of intestinal fistula and the surgical procedures which may be used to close them.

Two cases of localized phlegmonous enteritis presenting symptoms of acute intestinal obstruction, are reported by Irwin and McDonald[47]. Both cases terminated fatally from a rapidly spreading peritonitis; pathological examination suggested an autogenous infection, possibly resulting from local injury to the bowel wall.

Large Intestine. Carcinoma of the colon and rectum is discussed by Bloodgood[48], who reviews his experience with this disease and stresses the fact that ultimate cure depends chiefly upon the stage of the local malignant disease at the time operation is performed. He emphasizes the importance of preoperative treatment, and also of adequate intestinal drainage before resection. Operation in multiple stages is frequently useful. The author considers that if there are any symptoms of shock, blood transfusion should be carried out before this complication has become established. In resections, he advises a lateral anastomosis where it is possible to carry this out, as being a safer procedure than an end to end anastomosis.

In resection of the right side of the colon and for the establishment of an anastomosis between the terminal ileum and the transverse colon, Lahey[49] has utilized the Mikulicz type of operation. The article carries illustrations of his technique. In another paper he and Jordan[50] discuss the subject of carcinoma of the colon; the authors stress the fact that any alteration of the colonic function should be investigated early, to rule out the question of new growth, and conclude that early diagnosis must rest on x-ray pictures taken after opaque and air enemata. For resection of the colon, the Mikulicz plan is advocated as offering the safest method.

The diagnosis and treatment of carcinoma of the colon are also discussed by Young[51]. The author has devised special double intestinal clamps which have proved useful in carrying out aseptic anastomosis.

The complications associated with proctectomy, their prevention and treatment, are discussed by Whipple[52]. He advises the utilization of an indwelling catheter in the bladder for the first four or five days after operation. Since intestinal obstruction is a frequent complication of this operation, he advises attention to detail, in order to secure a smooth repair of the pelvic floor, and a careful attachment of the mesosigmoid to the lateral peritoneal wall to prevent the occurrence of a hernia to the left of the colostomy opening.

The details of management of permanent colostomies are dealt with in an article by Shedden[53]. This is a subject that is frequently neglected and yet is extremely important for the comfort and well-being of the patient. Shedden describes a type of colostomy dressing and retention cup which has proved satisfactory; and also gives suggestions for regulating the bowels and for technique which the patient can carry out in irrigating the large intestine through a colostomy opening.

The indications for the use of ileostomy for chronic ulcerative colitis, and the technique to be employed, are discussed by Bargen, Brown and Rankin[54]. The authors feel that ileostomy should not be performed until all other methods for treatment have failed; if it is performed it should be permanent. They consider that it has a definite field if performed for chronic complications of the disease.

Hyperplastic tuberculosis treated by surgical measures is the subject of a paper by Rankin and Major[55] based on a review of 65 cases. In 50 cases the mass had been resected and in 15 cases a short-circuiting operation performed. The authors point out that chronic intestinal

obstruction and fecal fistulae are frequently complications in this disease.

One hundred and twenty-three cases of so-called "colloid cancer" of the gastro-intestinal tract are discussed by Raiford[56]. He considers that the term "adenocarcinoma" or "mucoid carcinoma" is more accurately descriptive, and divides his cases into two groups, one of the true mucoid carcinomas arising as tumors of mucus-secreting cells; and the other, adenocarcinoma with degeneration. He considers the degree of mucus formation in adenocarcinoma roughly inversely proportional to the grade of malignancy; and also states that mucus deposits do not necessarily indicate malignancy.

Appendix. Numerous contributions to the study of acute appendicitis have appeared during the past year. Among these may be mentioned one by Clute[57], who discusses the factors he considers important in reducing the mortality from this disease. Among other points, Clute advises the use of spinal anesthesia and of a larger operative incision which permits the appendix to be removed while under constant inspection. Muller[58] also discusses the mortality in acute appendicitis and concludes that the immediate points of attack for the medical profession are the education of the public as to the dangers of the disease, and a further study of the best means of treating diffuse peritonitis.

An analysis of 2126 cases of acute appendicitis is presented by Sworn and Fitzgibbon[59]. Among other points made by the authors should be mentioned their views on those cases which are "too late for an early operation and too early for a late operation". They consider that all cases where no mass is palpable require immediate surgical treatment; whereas if a palpable mass is present, the best results are obtained by conservative treatment. In this latter group, however, there are certain cases which require immediate surgical treatment; the final decision must depend upon the judgment of the surgeon in charge.

Meckel's diverticulum in children is discussed by Hudson and Koplik[60] who base their remarks on a study of 32 cases. From a diagnostic point of view it is interesting that 17 of the cases showed hemorrhage from the bowel; and that in seven of these, the hemorrhage was the outstanding feature of the case.

ESOPHAGUS

Carcinoma of the esophagus is discussed by Gaetan and Emery[61] in a review of 61 cases. Dysphagia was the first symptom noted by 25 of the patients in this series; pain was the presenting symptom in 22 cases. The authors point out that either of these symptoms demands an x-ray examination, using an opaque medium, and also an esophagoscopy. Treatment of these patients was merely palliative.

MISCELLANEOUS

Tumors and cysts of the mesentery and omentum have been discussed in various articles[62, 63, 64, 65]; for lack of space, these rather rare conditions will not be reviewed.

Meleney, Olpp, Harvey and Zaytseff-Jern[66] have continued their studies on peritonitis. An article in the *Archives of Surgery* stresses the importance of the synergistic action of certain bacteria in producing a fatal peritonitis. Steinberg[67] has carried out experiments on a rapid method of protecting the peritoneum from peritonitis by the injection of colon bacillus vaccine. In general his work follows that of Rankin and others. Steinberg considers that the protection is not a true immunity, but that leucocytosis and phagocytosis are stimulated by the injections. Steinberg considers that protection could be obtained in four or five days after the immunizing dose.

Two interesting series of cases of gunshot wound of the abdomen are presented by Loria[68] and by Willis[69]. The mortality in the two series was 50 and 35 per cent, respectively. Hemorrhage is, of course, a very serious complication of most of these injuries and usually requires prompt transfusion. Willis advises not operating upon shotgun wounds where numerous birdshot have scattered throughout the abdomen; these small wounds of the intestines usually close without peritonitis, and it is hopeless to attempt to find the minute perforations. His article carries interesting photographs illustrating the lesions found.

A series of autopsies upon victims of automobile accidents, with special reference to rupture of an abdominal viscus is reported by Cooke[70]. Among the 210 cases autopsied 12 showed rupture of the intestines; it is interesting that in seven of these 12 cases there were no abrasions of the skin or other external evidence of injury to the abdomen.

Hernias following abdominal operations are often a very distressing complication; Bevan[71] presents a careful study of various types of abdominal incisions, and numerous illustrations of the anatomy of the abdominal wall, particularly of the nerve and blood supply.

The factor of food allergy in the differential diagnosis of patients with abdominal symptoms is discussed by Rowe[72]. The fact that allergic phenomena may produce acute abdominal symptoms was stressed many years ago by Osler. Rowe mentions especially that angioneurotic edema may cause severe pain and stimulate acute intestinal obstruction.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19481

AN UNUSUAL COMPLICATION OF PYONEPHROSIS

UROLOGICAL AND SURGICAL DEPARTMENTS

PRESENTATION OF CASE

DR. A. GORDON GAULD:* A seventy-one year old shoe worker entered the hospital with a complaint of pain in the right flank. He had been well until ten days before admission, when he became fatigued after a hard day's work chopping down a tree. The day after that for the first time he noticed that his urine was reddish in color. Two days later a doctor was called, who found a good many red cells and a few white cells in his urine. Five days later the patient felt a steady burning pain in the right flank radiating to the right groin. He had a slight frequency and pyuria at this time. The pain was considerably relieved by aspirin. The redness of the urine disappeared, but the urine remained cloudy until he entered the hospital. He had had no fever or chills until the day before admission, when his temperature rose to 101°.

Physical examination showed a fairly well developed and nourished man looking quite ill and complaining of pain in the right flank. The temperature was 102°, the white cell count 17,000. The abdomen was moderately distended but was not tender and no masses were felt. He had marked tenderness in the right costovertebral angle extending around to the right flank. Rectal examination was negative. The kidney was not felt on the right side. Cystoscopy was done the next day and a normal bladder found. The catheter passed easily to the right kidney and reddish urine was obtained. Examination of this urine showed only six white cells and very few red cells. Indigo carmine was injected and did not appear at all on the right side. Pyelograms showed a very definitely dilated and irregular kidney pelvis on the right. The ureters did not appear obstructed. An intravenous pyelogram showed essentially the same thing, with almost no excretion of dye on the right side.

A chest film showed a very high right diaphragm which moved with respiration.

The day following cystoscopy the temperature rose to 103°. The abdomen became somewhat more distended and the patient complained of dull pain, not localized, shifting from one side to the other. He vomited two or three times a day. On the third day the temperature was still about 103°. Tenderness had entirely disappeared from the right flank and had shifted to the lower abdomen.

A barium enema given at that time was unsatisfactory but did show an elongated rectum. Digital examination now showed a tender mass encircling the rectum.

On the sixth day operation was done. He improved temporarily for two or three days except that on the third day the temperature rose to 105° and he again complained of pain in the abdomen. He became irrational and unable to localize the pain. Drainage from the wound was profuse and the temperature continued to range from 102° to 103°. On the sixth or seventh day he failed very rapidly. On the eighth day he had chills one after another all day long. On the morning of the ninth day he died.

CLINICAL DISCUSSION

DR. GAULD: A diagnosis of perirectal abscess was made and the patient was transferred to the Surgical service. The perirectal abscess was drained and a great deal of pus was obtained.

Although it was realized that he had other foci of sepsis, we were absolutely unable to locate them.

DR. AUBREY O. HAMPTON: The intravenous pyelogram showed impaired function of the right kidney with a small area of calcification near the kidney pelvis. The intravenous pyelogram demonstrated the right kidney outline much better than the kidney pelvis or calices. The left kidney appeared normal.

The retrograde pyelogram gave us much more information. On the right side it showed a grossly dilated kidney pelvis and calices, with irregularity in outline which we thought indicated a badly infected kidney.

When we examined the chest we found an elevated right diaphragm which did move with respiration, but there was definite limitation in excursion. We thought we could explain the two abnormal findings by a perirenal infection, although we did see the kidney outline.

The barium enema reached the cecum and was promptly expelled. The patient complained of considerable pain in the rectum. The rectum as seen in the film is quite small, straight and smooth, and the mucosa is intact, a fairly characteristic picture of a large extrinsic annular mass.

DR. J. DELLINGER BARNEY: This case came into my hands at first. As you have heard, the symptoms were those of kidney infection, either perirenal or intrarenal. At the time when he

*Senior interne on the East Surgical service.

first came in there was nothing in his rectum to suggest pathology. The prostate was small and firm, with no induration or tenderness. As the catheter went into the right kidney, after going in for two to three centimeters it drained brownish granular material which looked like broken down blood clot. As it went up higher fairly clear urine drained. As it went into the kidney pelvis it struck a reservoir of this brown material. As you have heard, there was no function on that side. We injected indigo carmine and obtained a good function on the left side. The intravenous pyelogram showed marked destruction of the right kidney and the retrograde pyelogram also showed destruction of the kidney. The catheter on the left did not pass for more than four or five centimeters, though various catheters and various manipulations were tried. I have never been able to account for that. Autopsy showed a fairly normal kidney. All the symptoms and signs in the first few days pointed to renal or perirenal sepsis; but as these symptoms and signs shifted to his lower abdomen after my cystoscopic examination various things came to my mind. In the first place I wondered if the trouble which began after my examination might have been due to some injury I may have done to the ureter.

When the symptoms shifted and when we found this involvement around the rectum, being somewhat in a quandary as to what the patient had I asked Dr. Daniel Jones to see the case with me. At that time the perineum was more or less red and bulging and rectal examination showed a definite perirectal mass. I think Dr. Jones felt as I did, that the diagnosis was very obscure. At first sight it looked like a perirenal or cortical abscess of the kidney, but later on as more abdominal symptoms developed it became obvious that it was an ischiorectal abscess and he was transferred to Dr. Jones, who will tell you more about the case.

Dr. Daniel F. Jones: I saw this patient with Dr. Barney. It seemed to me that the history was perfectly typical of a perirenal infection, for these reasons. In the first place Dr. Barney said he had catheterized the ureter and had not obtained any pus. In the second place, the man had been doing hard work which was followed some time later by pain in his right flank. He had tenderness in the right flank at the time. If you look over the histories of perinephric abscess you will find that almost every patient has cut his lawn or chopped trees or done some heavy work just before the onset of pain. It is a very common occurrence.

On the basis of the very definite attack of pain in the right flank and the urine being clear I made a diagnosis of perinephric abscess. I saw nothing more of him for several days, when Dr. Barney said he had a swelling in the perineum. I saw him again and found his anus surrounded by an edematous reddened area. Rectal examination showed exactly what the x-ray does; that is, a thickened wall of the rectum with thickening outside narrowing the rectum to a small firm tube as far as the finger could reach. That is, the man had more than an ischiorectal abscess, he had a perirectal abscess, because it evidently came from above the levator muscles. When it was drained it was quite evident that it was a streptococcus infection from the appearance of the area. With the finger in the wound I could feel honeycomb tissue all over the pelvis in all directions. I could get my finger almost up to the top of the bladder without going outside this cavity.

This drained for some time and he seemed to be doing fairly well. Then he began to have chills. My feeling about chills is that the surgeon can do very little about them. When a man with a streptococcus infection begins to have chills that means, according to my way of thinking, that he has a fresh infection with each chill, and if a man has a fresh chill every day you cannot find all the foci and you may as well wait until you can find some place where you can drain and accomplish something. It does not pay to tear a man to pieces because he is having chills. You must have some idea as to where these fresh infections are before you can do anything in the way of surgery. The man went on getting worse and worse, the chills becoming more and more frequent until he died.

A Physician: Did you get a blood culture?

Dr. Gauld: Yes. It showed staphylococcus albus.

Dr. Tracy B. Mallory: Our blood culture at autopsy was colon bacillus.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Ischiorectal abscess.

ANATOMIC DIAGNOSES

Right pyelonephritis, pyonephrosis, hydroureter and pyoureter.
Perirectal abscess.
Acute hemorrhagic pancreatitis.
Fat necrosis of peritoneum, very slight.
Acute central necrosis of the liver.
Acute splenitis.
Acute bronchitis, right.
Pulmonary emphysema, slight.
Arteriosclerosis, slight coronary and aortic.
Operative wound, drainage of perirectal abscess.

Pathologic Discussion

Dr. Mallory: The terminal events here were again more than a little surprising. He had the huge perirectal abscess which Dr. Jones had drained, but it was apparently still extending upward. It went as high as the brim of the pelvis, but had not broken into the peritoneal cavity at any point.

There was also a marked infection of the kidney which showed innumerable small abscesses ranging from two to three millimeters in diameter,—a type of lesion which I should think could not possibly be effectively drained.

The final terminal event was apparently an acute hemorrhagic pancreatitis. The entire pancreas was floating in a lake of blood which was all enclosed within the capsule of the pancreas. The pancreatic tissue itself showed very little necrosis, although a few flecks of fat necrosis could be made out grossly and microscopically. I do not remember having seen before at autopsy such an acute hemorrhage in the pancreas as this. Ordinarily by the time the cases reach us the greater part of the pancreatic tissue has been totally destroyed. Dr. Jones saw the autopsy and told me the appearance was very much like that of the extremely acute pancreatitis which the surgeon sees when he operates within a few hours of the first symptoms.

It is obviously difficult to connect all these various things. The history would certainly suggest that the renal abscesses were the primary lesion. There was no evidence of any extension downward through the retroperitoneal tissues to the anal region. I have seen a pancreatitis do that and finally present in the pelvis and I think it is perfectly possible that a perirenal abscess might work downward and do the same thing; but there is no evidence that either of them had done it here. I think the perirectal abscess must be considered metastatic via the blood stream. Very probably the pancreatitis was also infectious in origin, although we cannot be very sure of that.

Have you any further ideas about the case, Dr. Jones?

DR. JONES: No. It is extremely complicated. Of course with perinephric abscess you could not very well have anything else with the findings; but when we came to see the kidney it was quite evident that it was not a perinephric abscess.

The pancreatitis is probably due to a metastatic infection such as occurs in infectious diseases occasionally. This type of infection usually causes an interstitial pancreatitis rather than a hemorrhagic pancreatitis.

I agree with you that the perirectal abscess must have been metastatic, because at the time of the autopsy you could find no communication between the right kidney and the pelvis. It was a very extensive and rapidly increasing abscess; that is, it must have been a very virulent organism and spread about in the soft tissues very rapidly, because the edema about the anus developed in a very short time.

DR. MALLORY: I neglected to say that there was no evidence of any injury to the ureter.

CASE 19482

ANGINAL PAIN FOLLOWING JAUNDICE IN A YOUNG MAN

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. NEIL L. P. CRONE:* *First admission.* A twenty-six year old single American born radio operator entered for the first time six weeks before his second admission with a complaint of jaundice of three weeks' duration.

Five weeks before entry the patient had a moderate head cold. Four weeks before entry he began to feel constantly fatigued, to notice anorexia and nausea and to vomit, usually about an hour after meals. Three weeks before entry jaundice appeared. During the subsequent period the nausea and vomiting gradually improved, though the jaundice steadily deepened. The patient, however, remained up and at work until the day before entry.

The family history was negative except for attacks of gall stone colic and a cholecystectomy in the patient's mother.

Nine years before admission the patient had gonorrhea, with inadequate treatment. Penile sore was denied. There were no skin manifestations. About five or eight years before admission the patient had a similar attack of jaundice lasting two to three weeks. Eight years before entry he was in a severe motorcycle accident which left him with a partially ankylosed knee. As a child he had measles, scarlet fever, whooping cough, diphtheria and two or three attacks of pneumonia.

Physical examination showed a moderately well developed and nourished man, deeply jaundiced, not appearing acutely ill. The teeth were markedly carious. The blood pressure was 130/80. The heart measurements were within normal limits, the sounds of normal quality, no murmurs. The lungs were negative. The abdomen was negative except for a palpable liver edge 3 centimeters below the right costal margin. The left knee showed a scar and limitation of flexion to forty-five degrees.

His temperature during the twenty-three days that he was in the hospital did not rise above 99.3°. The pulse varied from 75 to 100, averaging 85. The respirations were constant at 20.

Five urine examinations showed gravities ranging from 1.012 to 1.022, no albumin, sugar, or acetone bodies. Bile was present in large amounts in the first examination, gradually decreasing, and was absent in the final specimen. The blood hemoglobin was 80 per cent, the red count 4,680,000, the white count 10,850 to 6,800. The differential count was 68 polymorphonuclears, 30 lymphocytes, no mononuclears, 2 eosin-

*Formerly senior interne on the West Medical service. In his absence it has been necessary to publish Dr. Crone's part without his revision.

ophils, no basophils. Of five stool specimens examined the first two were clay colored, the third light brown, the last two normal in color. The Hinton test was strongly positive, the Wassermann weakly positive. A control examination in another laboratory showed both tests positive. The icteric index was 75 on two occasions. The van den Bergh gave a direct reaction on entry and a quantitative value of 22.5 milligrams, gradually dropping to 6.15 milligrams immediately before discharge. A liver function test on admission showed 100 per cent retention. It was not repeated.

X-ray examination: A flat plate of the gall-bladder (not a Graham test) was negative for stones.

On a high carbohydrate, low fat diet the patient slowly but steadily improved. The skin cleared somewhat, but the yellow color had not entirely disappeared and the liver was still enlarged on the twenty-third day, when he was discharged.

History of interval. He had felt quite well for two weeks. Then, four days before his return, he had a sudden very severe pain deep in the midsternal region. It made him feel short of breath and brought out a cold sweat upon his forehead. Soda and a hot water bag gave no relief. The pain persisted more or less constantly up to the time of entry. The slight jaundice present at his previous discharge remained unchanged in intensity.

Second admission. The patient reëntered eighteen days after discharge.

Physical examination showed no change except still greater enlargement of the liver, which was now felt almost at the level of the umbilicus. The spleen was now just palpable. The blood pressure was 140/65. The urine was free from bile. The stools were normal in color. The blood findings were unchanged. Three more Hinton and Wassermann reactions were all positive. The icteric index gradually dropped from 25 to 10. The van den Bergh was 1.5 to 2, direct reaction. X-ray examination showed no evidence of disease in the heart or chest. Electrocardiogram showed normal rhythm, rate 110, deeply inverted T_1 and T_2, rather low voltage.

He remained in the hospital one month, running for the first three weeks a temperature that reached 100° nearly every day. The pulse varied from 90 to 120. The respirations were fairly constant at 20. The chest pain slowly disappeared in the course of ten days, but a new pain appeared in the region of the fourth dorsal vertebra which gradually seemed to shift to an area behind the right scapula. Codein and pyramidon were given with relief from his pain. A syphilologist advised against arsenical treatment. Potassium iodide was started on the sixteenth day and continued until his discharge. One month after entry the patient went home, to continue on a high carbohydrate diet and rest.

History of interval. For the first three weeks after his discharge he felt well except for frequent attacks of palpitation. Five weeks before his reëntry on awakening in the morning he felt too weak to get up. During the next two days he frequently noticed cold sweats and twice had definite chilly sensations for a few hours. His physician, who was called in after three days, made a diagnosis of pneumonia, though the patient's temperature was never above 100° and he had no severe pain on breathing. During this period his most definite symptom was severe orthopnea, which gradually improved. Four weeks before reëntry his sputum became blood tinged and remained so for a week. The day before admission the sputum was again blood tinged. Three and a half weeks before reëntry moderate rather generalized pain was noted across the chest at the end of full inspiration. This gradually grew less severe. Three weeks before reëntry soreness was noted about the right ear, gradually shifting to the right temporomandibular joint and gradually becoming so severe that he could not open his jaw. Two and a half weeks before reëntry he became conscious of very forceful heart beats. This sensation continued until entry. During the week before entry three attacks of dizziness occurred.

Third admission, approximately two months after his previous discharge.

Physical examination showed definite orthopnea, marked pallor of the skin and mucous membranes and slight cyanosis. There were small palpable cervical, supraclavicular, epitrochlear and inguinal glands. The lungs showed dullness at both bases, greater on the right, with some diminution of breath sounds, spoken voice and whispered voice on the right. Tactile fremitus was normal. There were no râles. There was visible pulsation of the vessels of the neck, and capillary pulse in the lips and nail beds. The radial pulse was full and bounding. The apex beat was forceful, rapid, rate 128. A tremor could be felt with each beat. The heart was slightly enlarged. The sounds were of good quality, but somewhat obscured by murmurs. The pulmonic second sound was greater than the aortic second sound and much accentuated. In the second and third left intercostal spaces a systolic and a diastolic murmur were heard which were transmitted to the vessels of the neck and also to the apex. The blood pressure was 130/44. The liver was three fingerbreadths below the costal margin, tender, its edge smooth. The spleen descended two fingerbreadths with deep inspiration and was tender. There were tenderness and slight swelling of the right temporomandibular joint. The parotid area was not tender.

Laboratory examination of the urine showed the specific gravity 1.010 to 1.028, no albumin or sugar, bile on one occasion. Blood: hemoglobin 55 to 60 per cent. The red cell count was 3,200,000 to 4,100,000. The white cell count

was 9,150 to 16,400. The stools were normal in color. The Hinton and Wassermann reactions were positive. The icteric index was 8, the liver function 0-5 per cent retention. A gonococcus fixation test was positive. A prostatic smear showed numerous pus cells, nearly all of which contained intracellular Gram-negative diplococci. Three blood cultures were negative. An electrocardiogram on April 11 showed a sino-auricular tachycardia, rate 110, slightly inverted T_1 and T_2, tendency to low voltage. A second electrocardiogram on the twenty-ninth showed again a sino-auricular tachycardia, rate 120, inverted T_2 and T_3.

X-ray examinations of the temporomandibular joint showed widening of the joint space and anterior displacement of the articular surface of the mandible. The dislocation however was not complete. A heart plate showed considerable enlargement, most marked across the base, in the region of the auricles. The shadow of the left auricle was particularly prominent. The left bronchus appeared elevated. The pulmonary conus was visible. There was marked increase in the hilus shadows on both sides with some mottling in the mid and lower lung fields extending well out toward the periphery. The aorta appeared normal. Comparison with films taken in January showed marked change in the size and shape of the heart and also in the size and density of the hilus shadows. The findings were interpreted as rheumatic heart disease with pulmonary congestion. Another chest plate taken on June 2 showed an evident increase in the size of the heart since the last examination. The shape still suggested mitral disease. The aortic knob was not visible. There was prominence in the region of both auricles. The hilus shadows and larger lung markings were still more prominent. The findings were interpreted as mitral stenosis and congestion of the lung.

During the two months that the patient remained in the hospital he was treated with digitalis, Blaud's pills and potassium iodide. The patient went rather rapidly downhill. Attacks of air hunger were frequent. His appetite was often poor, and medication occasionally had to be withdrawn because of nausea. He had several transient acute upper respiratory infections. His temperature, which had ranged about 99° at admission, rose with some of these attacks to 100° and 101°. The physical findings did not change until May 29, when a presystolic murmur and thrill were noted at the apex, thought to be an Austin Flint murmur. Towards the end of May the vomiting became more and more frequent, the liver appeared to grow larger and more tender, vomiting and hiccoughing became almost continuous. On June 6 the patient suddenly sat up in bed gasping for breath, then dropped back dead as the nurse started to hand him some smelling salts.

DIFFERENTIAL DIAGNOSIS

Dr. James H. Means: Here we have a young man entering the hospital three times in less than five months and dying there about eight months after the apparent onset of his illness.

The picture on the first entry is that of an acute parenchymatous liver disease, indistinguishable from acute infections (so-called catarrhal) jaundice. On the second entry the picture is that of an acute abdominal condition characterized chiefly by severe upper abdominal pain and low grade fever. The picture on the final entry is that of a fulminating type of heart disease with rapid development of aortic valve involvement, left ventricular failure and sudden death.

From the point of view of diagnosis it is very significant that on neither the first nor the second entry was any evidence obtained of any sort of lesion of the heart. Not only were there no symptoms pointing to the heart, unless the pain on the second might possibly be coronary, but both physical examination and x-ray showed complete cardiac normality. This narrows the diagnostic search considerably, for there are not many sorts of heart disease that can proceed at the pace which the facts here indicate. Indeed, I can think of nothing save malignant endocarditis, syphilis or some complication of coronary thrombosis. It is our duty to attempt to explain the entire picture by one etiological diagnosis. A bacterial infection might conceivably produce first a hepatitis, later an ulcerative endocarditis. Syphilis might involve both heart and liver with rapidly progressive lesions. I can think of no other etiologies which would account for all we have here. The coronary possibility is, I think, unlikely. His age is against it, the behavior of the blood pressure is against it, and as a diagnosis it fails to account for the development of aortic valve disease and the type of liver lesion that seems to be present. I feel that we can dismiss it.

Gonococcus infection is to be thought of; indeed, apparently he has it. There is the positive history of urethritis, positive gonococcus fixation test and prostatic smear showing organisms with gonococcus morphology and staining properties. The acute arthritis of his jaw, noted on the third entry, could easily be Neisserian. The gonococcus could also produce a fulminating endocarditis of the sort which may be present here. I do not see how, however, by the wildest stretch of the imagination the gonococcus could be blamed for this lesion, and for that reason and because of the extreme rarity of gonococcus endocarditis I feel justified in drop-

ping a gonococcus infection as the primary etiology.

There are several things present that smack of syphilis: Hinton and Wassermann always positive, development of a generalized lymph adenopathy and location of the heart lesion in the aortic valve.

I should be interested to know with what diagnosis the patient was discharged the first time. It seems to me that the symptoms, signs and laboratory work could all be explained on the basis of catarrhal jaundice. The marked dye retention, combined with obstructive jaundice, indicates a diffuse liver lesion with intrahepatic obstruction. The course of the disease is consistent with catarrhal jaundice. It is to be noted, however, that he was not quite well on leaving the hospital. Arsphenamin jaundice is to be thought of, but there is no story that he had had that drug. The beginning of an infectious cirrhosis is a possibility.

The picture on the second entry, that of epigastric pain and considerable increase in the size of the liver, practically proves that a diffuse liver disease of more formidable sort than catarrhal jaundice is present. There is no cardiac congestive failure to explain the liver and of course neoplasm is not in the least suggested. I see no escape, at this time, from some kind of cirrhosis, which could be syphilitic or other. The pain could be quite well explained on the basis of cirrhosis. We have seen it not infrequently. It is of diagnostic importance that he was started on potassium iodide and that coincident with the taking of this there was some improvement; that also smacks of syphilis. Of course, they were afraid to give arsenic. Who wouldn't be?

On the third entry he turns up with a brand new heart lesion. The double murmur and Corrigan pulse seem to me to point unmistakably to the aortic valve. Although the x-ray people suggest rheumatic heart disease, I feel that this is most unlikely. My interpretation would be that with rapid anatomical change in the heart, great strain had rather suddenly been thrown on the organ with but little time for hypertrophy and consequent left ventricular failure (note the attack of air hunger) and pulmonary congestion. At the same time the liver lesion is progressing without any sufficient evidence of right sided failure to explain it. The liver disease seems certainly primary, not secondary to the heart. All this hangs together on a syphilitic basis better than on any other. If he had a malignant bacterial endocarditis, I cannot understand why the organism could not be easily found on blood culture, also why there were no embolic phenomena.

DR. MEANS' DIAGNOSIS

Diffuse syphilis of the liver (syphilitic cirrhosis).

Syphilitic heart disease—with chiefly aortic valve involvement.

Arthritis of the jaw, probably gonorrheal.

Chronic gonorrheal prostatitis.

Sudden death, occasioned chiefly by the heart lesion.

CLINICAL DISCUSSION

DR. CRONE: The diagnosis at discharge was luetic heart disease, lues of the aorta and lues of the liver.

DR. AUBREY O. HAMPTON: When our first examination was made we said the heart was normal in size and the cardiothoracic ratio was within normal limits. We did not perhaps pay as much attention to the size of the pulmonary blood vessels as we should have done, and possibly if we had examined the heart in the oblique view with barium in the esophagus we should have found prominence of the left auricle; since increase in the pulmonary blood pressure is obvious in the anteroposterior view it should be obvious in the oblique view. There is nothing else that I can see to suggest heart disease.

On the second examination, three months later, the heart has changed from 11.6 centimeters in the transverse diameter to 12.9 centimeters, an increase of a centimeter, and there is definite evidence of passive congestion in the lung fields. It is even possible that that area of dullness thought to be due to edema represents an area of consolidation on the left side. The left bronchus is well seen here, not so well there. It seems to have come up a little, as we see in the presence of a dilated left auricle.

In the third film the transverse diameter has increased three centimeters within one month. We have been believing that a sudden increase in the size of the heart is usually due to pericardial effusion. We have recently discovered that myocardial failure or disease will do almost the same thing.

DR. CHESTER M. JONES: I saw this patient I think four times. I saw him at his first entry to the ward, at the second entry, and then in the Gastro-intestinal Clinic a day or so before he came into the ward, and I urged him to come in a third time.

There are one or two things that I recall about the case that interest me. I am not at all familiar with intense jaundice in association with syphilis except in the late stage of syphilitic cirrhosis. He certainly did not suggest that when he came in. The clinical picture was not unlike an acute catarrhal jaundice. It seemed to me however that he was more sick and that the jaundice persisted longer than one would expect in catarrhal jaundice alone, and I remember that I made a note at the time that I thought he had something else besides catarrhal jaundice.

DR. TRACY B. MALLORY: Another point is that he had already had one attack of catarrhal jaundice five years previously.

DR. JONES: That being true, he had a basis for further liver damage if one accepts the conviction that many people hold now that catarrhal jaundice is essentially acute yellow atrophy in greater or less degree.

The second thing that stood out—and I think we made a mistake in not paying attention to it—was the strongly positive Hinton on the first admission. I was taught that in the presence of deep jaundice one has a right to question and disregard serological evidence of syphilis. My own experience however is that it is a very rare thing to get a positive Wassermann or Hinton reaction with deep jaundice unless there is syphilis present. Whether that is true I do not know, but I do know that I am going to pay more attention to positive Hinton reactions with jaundice. It was felt that antisyphilitic treatment should not be pushed at this time because of the evidence of the liver disease and he was discharged without any attempt to treat his syphilis.

He came in later with a story of peculiar substernal pain. He had a much larger liver at that time without any evidence of jaundice, or with very slight jaundice. I think that I saw him once then and my recollection is that I felt that there might be a mediastinal or a perihepatic process which was responsible for his pain and fever. The fever persisted for a month, starting out in high swings and gradually coming down to a level. With rest in bed his pain disappeared. I did not think much about his heart at the time, and at that time antisyphilitic treatment was started cautiously. He was sent home.

The next time I saw him was in a sense accidental. He was discharged to the Gastro-intestinal Clinic. When he came in he was in extremely bad shape, very pale, weak, cyanotic, and obviously suffering from symptoms of heart failure. I examined him, and in listening to the heart I was amazed to find perfectly clear cut signs of aortic insufficiency, if one can make a diagnosis on murmurs alone. I think there was no record of a diastolic murmur at the second entry. He had signs of heart failure which were very striking. He was orthopneic and cyanotic. I thought at that time that the enlargement of the liver was in part due to cardiac failure and in part to his previous condition. I wondered if he had not had a rapid syphilitic involvement of the aorta and whether the whole picture could be due to unusually rapid syphilitic manifestations involving both the liver and later the heart.

His subsequent downhill course was certainly fundamentally that of cardiac failure. He did not respond to treatment at all. I should expect that there might be evidence of previous atrophy and repair in the liver to some extent. I believe he probably had cirrhosis, exaggerated by his cardiac failure.

A PHYSICIAN: Was there any evidence of gonococcal arthritis?

DR. MALLORY: No, unless the lesion of the temporomandibular joint was evidence of it.

Dr. Cabot, have you any feeling as to the diagnosis here?

DR. RICHARD C. CABOT: The thing that strikes me is the extraordinarily rapid enlargement of the heart as shown in these x-ray films. So far as my experience goes it is very unusual to get a heart as large as that shown in the third film so very short a time after the small, indeed normal dimensions shown in the first. I have nothing to say as to explanation, except that it seems very queer. I have never seen it before.

The same remarks hold true of aortic regurgitation due to syphilis. I have never seen it develop quickly. It ought to be and is usually a slowly developing thing.

Another point that ought to be balanced here is the question between rheumatic and syphilitic cause. If the disease is rheumatic we should be much more likely to get something in the mitral. As I understand it, Dr. Hampton said the x-ray was suggestive of mitral disease.

DR. HAMPTON: I think we leaned very strongly towards mitral disease.

DR. CABOT: A mitral lesion is never due to syphilis. If there is a mitral lesion I think we ought to suppose the commonest alternative, which is a rheumatic lesion. I do not see how this can be ruled out.

So far we have heard nothing about cardiac infarct. He certainly had suggestive pain, and the electrocardiogram suggested it. On the other hand I have been struck all my life by the way in which aortic regurgitation of syphilitic origin causes epigastric and substernal pain which cannot be accounted for by infarction, although it is very much like that due to infarction. I should think infarction ought to be considered here, but need not be present.

I shall be interested to see what the liver shows. It certainly is not the picture of liver syphilis unless it is the syphilitic form of cirrhosis, which I suppose we almost never recognize clinically.

DR. MALLORY: We had last year one case of what we believed was acute syphilitic hepatitis with jaundice, the patient dying, as I remember, of acute middle ear infection and meningitis, which allowed us to see the hepatitis in an acute stage. I doubt if a patient ever dies solely of syphilitic hepatitis in the acute stage. I think that jaundice is not extremely rare in the acute stage, although it usually runs a benign course even without treatment.

DR. JONES: This was very intense jaundice. Is it not usually a rather mild affair?

DR. MALLORY: In the statistics of acute yellow atrophy, the disease appears to be unduly frequent in luetics, but of course arsphenamin therapy may explain the findings.

DR. CABOT: May I ask Dr. Hampton about

the point he brought out about the left bronchus? What is the relation, radiologically speaking, of the left bronchus to the auricle in mitral disease?

DR. HAMPTON: When the left auricle is dilated the left main bronchus is pushed upward and in some cases the bronchus to the lower lobe is obstructed by pressure. That displacement of the bronchus upward which we see in this film is not so marked as in chronic mitral disease. It is just suggestive. The thing upon which we chiefly based our diagnosis is the fairly positive evidence of increase in the pulmonary pressure without much left ventricular enlargement. You see the enlargement is in all diameters and across the base. You can hardly see the aorta; therefore it is not very large.

CLINICAL DIAGNOSIS

Luetic heart disease.

ANATOMIC DIAGNOSES

Myocarditis, possibly luetic, with failure.
Luetic aortitis with rupture of the attachment of one of the cusps.
Small aneurysm of the sinus of Valsalva.
Pulmonary congestion.
Hydropericardium.
Cirrhosis of the liver, unclassified.

PATHOLOGIC DISCUSSION

DR. MALLORY: The extraordinary rapidity of the development of the cardiac symptoms and findings is certainly the outstanding feature of this case. I think all of us would certainly agree that we have never seen a case of syphilis progress at this rate, yet I believe that is what he had and all that he had.

The heart was only slightly hypertrophied. Of course the duration of his cardiac symptoms was relatively short. The heart was, however, considerably dilated. The only valvular abnormality was in the aortic valve. That was quite different from anything that I have ever seen. One of the cusps of the aortic valve was actually torn off the aorta. At the point where that rupture had occurred there was a scar, and behind that the sinus of Valsalva made a large pocket which could be described as a small aneurysm immediately behind the point at which the valve cusp had ruptured. I have seen that sort of thing in acute endocarditis but never in syphilis. In this case however there was no trace of a vegetation on any of the valves, so that we can add anatomical evidence to the clinical absence of positive blood cultures in ruling out acute endocarditis entirely.

The liver, which at all examinations had been considered very large, was slightly smaller than normal, weighing 1500 grams. It was very deep colored and had quite a granular surface. When we tried to break it or cut it, it was much tougher than normal. There was unquestionably a considerable degree of cirrhosis present, distinctly more than we can account for by the so-called cardiac cirrhosis. With chronic passive congestion, I think almost solely in rheumatic heart disease of many years' duration, we can get a contracted, cirrhotic scarred liver. Even in the most extreme cases of that sort, however, the degree of cirrhosis is still a relatively slight one. Here there was a marked cirrhosis and marked passive congestion simultaneously.

The coronary arteries were entirely negative. There were no other gross findings of much significance.

DR. HAMPTON: I did not understand what you said about the cusp overlapping the coronary artery orifice.

DR. MALLORY: Where two of the aortic cusps normally come together at the commissure, their attachment to the aorta was completely severed, though they remained slightly adherent to each other, so that we had a sort of bicuspid valve, one huge cusp flapping, practically without attachment, and a smaller cusp opposed to it. The lesion however appeared plainly acquired, and was not at all of the congenital type.

DR. HAMPTON: Where was the coronary artery?

DR. MALLORY: It was in behind this area.

DR. CABOT: The coronary was not obstructed at all?

DR. MALLORY: No.

The first section here is from the myocardium. The changes are rather slight, so that I shall be able to demonstrate only a portion of them. Around all the blood vessels there is a very considerable increase in fibrous tissue. There are numerous areas where occasional muscle fibers are undergoing a hyalin degeneration, and in those areas there is a certain amount of infiltration of lymphocytes and plasma cells. An occasional coronary branch shows marked thickening of the intima with fibrous tissue. The process is not an active one, but it can be the end result of an endarteritis and is perfectly consistent with a luetic lesion, so that I think we have the right to say there is a progressive myocarditis in this case.

The aorta shows typical very marked collections of lymphocytes around the vessels of the adventitia, and in some places infiltration extending into the media with a tendency towards vascularization of the media.

The lesion is reasonably specific for lues. It is not impossible that the same picture might be produced in a rheumatic aortitis, but it seems to me that if that were the case we should have much more definite evidence of rheumatism elsewhere in the body than we have found.

This final section is from the liver. In the center of the field is a portal area with increase in fibrous tissue and increase in bile ducts.

There is a very narrow zone of liver tissue on each side of it, and in the center of the lobules the liver cells have disappeared. The picture is in part that of passive congestion, but also of considerably more definite cirrhosis than can be so explained. It fits the toxic type of cirrhosis which Dr. Jones was predicting, but it would be difficult to rule out syphilis.

A STUDENT: I should like to ask if at any time there was a whistling or a singing murmur heard?

DR. MALLORY: I think not. It was not noted in the record.

DR. CABOT: How do you account for the substernal pain?

DR. MALLORY: A considerable number of cases of aortic valve involvement have it, and so far as I have been able to make out, it makes very little difference as to the type of involvement. Rheumatic, luetic, and calcified senile stenosis—all three show it.

A PHYSICIAN: You do not think it is related to the dilatation of the aorta at all?

DR. MALLORY: I certainly cannot rule that out.

A PHYSICIAN: Was the aorta distinctly enlarged?

DR. MALLORY: There was one localized pocket. The rest was not above the normal diameter.

DR. JONES: The strange thing was that in the second entry, when he had his most intense pain,—he came in on account of pain and fever,—he showed absolutely no evidence of aortic disease. It was not until the third entry that one could identify such a condition.

A SURGEON: Could it be due to a stretching of the pericardium, this rather abrupt enlargement of the aorta?

DR. MALLORY: I should think it might be. Are there any pain fibers actually in the aorta itself? The time at which the pain began would fit very well with the time that process must have started to erode his aortic wall, although certainly the usual case of syphilis does not give any anginal story.

DR. HAMPTON: We have had one similar case at the Baker where the diagnosis was also rheumatic heart disease and there was quite the same picture, and the patient died of acute luetic aortitis, and was explored for pain.

A PHYSICIAN: Was there a ruptured valve?

DR. HAMPTON: No. In that case the process was largely in the lower aorta, most marked in the abdominal aorta. The pain had been abdominal.

DR. JAMES C. WHITE: Dr. Paul White assembled three or four cases of aortic regurgitation in young people all of whom showed a destroyed aortic valve with very marked regurgitation, and he feels that most of the pain is caused by low pressure in the coronaries during diastole. The coronary arteries themselves are normal, but insufficient blood flows into them. This case illustrated that very markedly.

DR. JONES: May I ask Dr. Cabot one question? What is your experience with a positive Wassermann with jaundice?

DR. CABOT: Just like yours. I have recently seen a number of cases where I have disregarded it wrongly, and I have come to exactly the same conclusion you have, that it should not be disregarded any longer.

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FRACASTORO AND HIS FAMOUS POEM

IN the year 1521, Girolamo Fracastoro, Italian physician, epidemiologist and poet, spent the summer at his country home at Caffi, a few miles from his native city of Verona. Beautifully situated at the foot of Monte Baldo and overlooking Lago di Garda, Fracastoro's villa offered not only a place for reflection and meditation on the unsettled state of Italy, but it also afforded a retreat from the plague which was then raging through Verona, a kind of contagious distemper long of interest to this physician. Here, at least, the violent and sudden winds of Garda cleared the air of those "minute bodies" which the learned doctor suspected were the cause and means of transmission of this infection. And in these beautiful Lombardian surroundings Fracastoro wrote his poem, *Syphilis sive Morbvs Gallicvs*, in which one finds not only a description of the disease as seen in its epidemic form, but the name syphilis occurring for the first time in medical literature. *Syphilis* was regarded by Osler as the greatest of all medical poems.

Fracastoro sent his poem to his friend, the scholar Pietro Bembo, secretary to Pope Leo X, who is said to have revised the original draft. For some reason the verses were not published until nine years later. In the meantime Bembo's patron, Leo X, had died (December, 1521) and it was not until 1529 that Bembo became historiographer to his native city of Venice and librarian of St. Mark's. Whatever may have been the reason for the long delay, a point which has never been clearly explained, the book was issued in an attractive form without imprint in 1530. It has been found that the volume was from the celebrated press of Stefano Nicolini da Sabbio of Verona. Since then it has been translated into at least six languages and there have been issued over one hundred separate editions.

Recently investigations have been undertaken toward making a complete bibliography of this medical classic and a preliminary list of known editions has been issued*. It is hoped that scholars throughout the world will assist in this important piece of historical research, which is being undertaken by Dr. Leona Baumgartner and Dr. John F. Fulton of the Yale Medical School.

*Handlist of Editions of the Poem *Syphilis sive Morbvs Gallicvs*. Oxford, University Press, John Johnson. Privately printed. 1933, viii, 20 pp.

A CODE IN PLANS FOR THE CARE OF INDUSTRIAL WORKERS

AT the recent meeting of the American College of Surgeons in Chicago Dr. Frederick A. Besley announced that Dr. Franklin A. Martin and he had had a meeting with Mr. W. L. Allen the Deputy Administrator of the National Recovery Administration and that he had agreed to the principle of their suggestions for a code to govern the N. R. A. in plans for the medical and surgical care of industrial workers. The provisions of this code are as follows:

1. All industrial medical clinics shall be requested or required to conform to the standard for industrial clinics as it has been established by the American College of Surgeons (or organized medicine).
2. It shall be requested or required of employers that all new employes shall be given a pre-employment examination and records kept of this examination and that a health audit be made of all employes, old or new, at least once a year. This examination to be made by the physician and surgeon employed by the given industry.
3. That industry further be requested or required to establish and maintain a pathological and x-ray laboratory. If

the industry employs one or more thousand this is to be an individual laboratory for such a plan, but in smaller industries a common or joint laboratory may be maintained in communities.

4. Any physical defect revealed at these examinations shall be cared for by the industry in so far as they come within the scope of the workmen's compensation law of any given State.
5. If physical defects are discovered by these examinations which do not come within the scope of the workmen's compensation law, then the patient should be instructed to consult his or her family physician for treatment and the physician of the patient should be furnished the facilities and records of the industrial laboratories, and this service should be rendered at a minimum cost.
6. In so far as practical, the facilities of the laboratories should be available to the family physician for service to the family of the employe, and the medical department of the industry should correlate with him in arriving at a diagnosis.
7. Insist that industry utilize hospitals which are equipped with proper facilities and standardized. These hospitals should be used, as far as feasible, as health centres where periodic health examinations may be made.

At first thought this program might seem to be too burdensome to industry to allow its being carried out. However, there is reason to believe that it would not be burdensome at all. Besides the benefits to the individual employe, the cost to industry would be greatly reduced in comparison to the unknown but very large indirect costs under the present hit or miss system which furnishes inadequately a large part of the accident and sickness prevention work that is being done in most industries. Even the adequacy of treatment given the injured at present is open to not a little question. Can a group of people be given the most economical service under the conditions in which the industrial accident cases are treated at present in Boston? When hospitalization is needed the great majority of cases are sent either to one of the great public hospitals of the city where about half of the hospital costs are met by the taxpayer or by private charity and the medical costs are met by the kindness of the surgical staff, or to some distinctly second-rate hospital not approved as a hospital by the American College of Surgeons and where the care given and standards of practice are frequently archaic.

The American College of Surgeons should be congratulated on this forward looking program.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MILLER, JAMES RAGLAN. A.B., M.D. Johns Hopkins University School of Medicine 1911. F.A.C.S. Assistant Obstetrician and Gynecologist, Hartford Hospital. Gynecologist, Hartford Municipal Hospital. Consulting Gynecologist, Bristol (Connecticut) Memorial Hospital, Windham Community Memorial Hospital, Willimantic, and Neuropsychiatric Institute and Hospital of the Hartford Retreat. His subject is "Spontaneous Rupture of the Stomach During Labor." Page 1085. Address: 179 Allyn Street, Hartford, Conn.

GARGILL, SAMUEL L. M.D. Tufts College Medical School 1925. Assistant in Medicine, Harvard Medical School and Associate Physician and Physician to the Thyroid Clinic, Beth Israel Hospital, Boston. His subject is "The Use of Sodium Dehydrocholate as a Clinical Test of the Velocity of Blood Flow." Page 1089. Address: 485 Commonwealth Avenue, Boston.

HOFFMAN, FREDERICK L. LL.D. Tulane University, 1911. Consulting Statistician Prudential Insurance Company. Member, American Cancer Research Association. Director, American Society for the Control of Cancer. His subject is "Cancer of the Male Generative Organs." Page 1093. Address: Wellesley Hills, Mass.

PARSONS, LANGDON. A.B., M.D. Harvard University Medical School 1927. Assistant in Surgery, Massachusetts General Hospital. Surgeon to Outpatients, Huntington Memorial Hospital. Assistant Visiting Surgeon, Pondville Hospital. Assistant in Surgery, Harvard Medical School. His subject is "The Operative Curability of Carcinoma of the Stomach." Page 1096. Address: 264 Beacon Street, Boston.

CARR, F. B. A.B., M.D. Harvard University Medical School 1927. Assistant Cardiologist, Worcester City Hospital. His subject is "Functional Bundle Branch Block." Page 1101. Address: 27 Elm Street, Worcester, Mass.

MACOMBER, DONALD. A.B., M.D. Harvard University Medical School 1909. F.A.C.S. Research Fellow in Obstetrics, Harvard Medical School. His subject is "Studies of Reproduction in the Rat. I. The Effect of Changes in the Protein upon Fertility, Pregnancy and Lactation." Page 1105. Address: 321 Dartmouth Street, Boston.

WORCESTER, ALFRED. A.B., A.M., Sc.D., M.D. Harvard University Medical School 1883. Professor of Hygiene, Harvard University. His

subject is "The Boston Lying-In Hospital Fifty Years Ago." Page 1109. Address: 314 Bacon Street, Waltham, Mass.

McIver, Monroe A. A.B., M.D. Harvard University Medical School 1917. F.A.C.S. Surgeon-In-Chief, Mary Imogene Bassett Hospital, Cooperstown, New York. His subject is "Review of Gastro-Intestinal Surgery in 1932." Page 1113. Address: Mary Imogene Bassett Hospital, Cooperstown, N. Y.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

Problem of Tuberculosis in the Adolescent*

BY DONALD S. KING, M.D.

To understand the problem of tuberculosis in the adolescent, one must understand something about the development of tuberculosis before and after adolescence. The main point to be grasped is that the lungs react differently to a first infection with the germs of tuberculosis than they do to a second or later infection. When a moderate number of tubercle bacilli are taken into the lungs for the first time, the protective forces of the body are able to combat the infection, the infected person has few or no symptoms, and the tubercle bacilli are finally imprisoned in the glands located around the larger bronchial tubes. This type of tuberculosis is called the first infection type or since the first infection takes place usually in childhood, the childhood type. Following such a first infection the average child enjoys a period of good health. Lime salts are gradually deposited in the infected glands and the x-ray film shows these glands to be calcified. These calcified glands have been the subject of much study. It was long believed that if a gland contained calcium, the tuberculous infection was completely healed, all the tubercle bacilli had been killed and the war was over. Many authorities have gone even farther and maintained that a calcified gland meant that the body had not only overcome the first infection, but in so doing had built up a protection or immunity against any subsequent infection.

Further investigation has, however, shaken this belief. In the first place, living tubercle bacilli have been found in glands that have been calcified for ten, twenty, or even thirty years and, in the second place, repeated x-ray examinations of school children show that those with calcified glands are more apt to develop tuberculosis in later life than those who do not have this evidence of previous tuberculous infection. This important point is far from being settled but several recent studies give support to the belief that an infection with tubercle bacilli in childhood increases rather than decreases the danger of tuberculosis later in life. So much for the first infection or childhood type of tuberculosis.

After a period of quiet so far as activity in the glands is concerned and after a possible short period of immunity to fresh infection from the outside, many people develop tuberculous infection not in the glands but in the lung tissue itself. In some cases this infection is so active that portions of the lung soften and as this liquid material is coughed up, holes or cavities are left in the lung. In other cases, the patient's resistance is better and the infected area is gradually healed and replaced by scar tissue. In either case, this type of lung or pulmonary tuberculosis is called the adult type since it usually occurs in later life. With present knowledge it is impossible to say whether the adult type of infection is more often caused by the escape of tubercle bacilli from bronchial glands than by an infection with new bacilli coming in from outside.

So much for the two types of infection, the childhood type which is for the most part glandular and the adult type which is the true pulmonary form. Now, what about the adolescent? The names at present applied to the two forms of tuberculosis are unfortunate for, if we use these terms, the adolescent may have either the childhood type, if he receives his first infection during the adolescent age, or the adult type if in adolescence, a later infection takes place. In the cities, at any rate, a large proportion of the children are slightly infected with tuberculosis during childhood and a fresh infection in the adolescent period usually leads to the adult form.

There are three important facts about tuberculosis at this age. (1) The infection is very apt to develop insidiously and by the time the child shows symptoms of disease such as cough, expectoration, fever, and loss of weight, the diseased process may be advanced and cure difficult. Cases have been reported in which the x-rays showed a gradually developing lung process for seven years before the child showed any symptoms of infection.

(2) Perhaps because it is usually discovered late, or perhaps because the adolescent lacks resistance to tuberculous infection, the death rate among those developing the adult type of tuberculosis is extraordinarily high. In 1900, the mortality tables for all ages showed that tuberculosis caused more deaths than any other disease. In 1930, again considering all ages, tuberculosis ranked seventh as a cause of death. However, if we consider the ages between ten and thirty-five, tuberculosis still ranks far above any other disease. The seriousness of the adult type of tuberculosis can be seen from the fact that out of 110 children found by the Massachusetts Department of Public Health in its school clinics to have this form of disease, 23 per cent were dead within three to seven years.

*Station WBZ, October 20, 1933, 4:30 P.M.

(3) The disease is more frequent and the death rate much higher in adolescent girls than in boys of the same age. The Massachusetts study has shown almost three times as many girls as boys with this type of tuberculosis. Furthermore, a study of the death rates shows that one out of every three young women, who die between the ages of fifteen and thirty, dies of tuberculosis.

Such figures are rather terrifying and lead at once to the problem of prevention. Here there are two questions: first, how shall we prevent a tuberculosis patient from giving his germ to an uninfected person (a problem too big for discussion here); and secondly, how can we make an early diagnosis of the adult type of tuberculosis?

Early cases are most often found by careful study of certain groups of children. These groups are first, those with any suspicious symptoms of lung trouble; secondly, those known to have had the childhood type of tuberculosis; thirdly, those who have had known contact with persons coughing up tubercle bacilli; and fourthly, those without a previous history of infection or exposure but with a positive tuberculin skin test, the so-called von Pirquet or Mantoux test. This test is simple, harmless, and accurate and shows whether a child has at any time been infected with tubercle bacilli, though it does not show whether the infection has been walled off in the glands or has already spread to the lungs. However, if the test is carefully done and is negative, there is only a small chance that the child has ever been infected with tuberculosis, and still less chance that he has active disease. All children with a positive tuberculin test should have an x-ray film taken of the lungs since physical examination is not sufficient to rule out even a serious lung involvement. So much depends upon the x-ray that it is very important that the film be carefully taken and interpreted by a person of wide experience. Even if the first x-ray is negative, it is often necessary to repeat the examination, perhaps several times.

Once the diagnosis of the adult type of tuberculosis has been made, the child should be followed with extreme care. In a few of these cases careful study will show that the infection is already arrested and some of these children may be allowed to continue with their ordinary life, providing frequent x-ray examinations are made. Usually, however, if the x-ray evidence is certain, even though the patient may have very slight or no symptoms, a long period of bed rest is indicated.

Often, however, even prolonged bed rest has not stopped the progress of the disease. Fortunately for these cases, there have been recent improvements in the treatment of tuberculosis and where bed rest is not effective, some of the modern surgical methods should be carefully considered. In many instances, these methods have undoubtedly saved lives which otherwise would have been lost.

To summarize: every effort should be made by tuberculin skin tests and x-ray to establish an early diagnosis of the so-called adult form of tuberculosis in the adolescent; if a carefully interpreted x-ray shows such a condition it must be taken seriously even though symptoms of infection are absent.

OBITUARY

RESOLUTIONS ON THE DEATH OF SAMUEL JOHN HARTMERE

Samuel John Hartmere, who died at the Boston City Hospital, November 1, following a heart attack, was born in East Boston, June 5, 1895.

He attended the Boston Schools and also Hobart College, Geneva, New York, and graduated from Tufts College Medical School in 1929 and from the Aural Service of the Boston City Hospital. He later was appointed to the Aural Visiting Staff of the Boston City Hospital and also the Carney Hospital.

He was a member of the Winthrop Lodge of Elks, Winthrop Council Knights of Columbus, Winthrop Kiwanis Club and the University Club of Boston.

He was greatly handicapped by his physical disability yet he was always willing to give his time to the ill and needy. He was of a cheerful disposition and well liked by his associates.

In his passing the Carney Hospital has lost a willing worker and a capable surgeon.

He is survived by his mother, a sister and two brothers. To his family we extend our sincere sympathy.

Signed:
FRANCIS G. MINITER, M.D.

November 20, 1933.

NOTICES

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

On Thursday, December 7, in the amphitheatre of the Peter Bent Brigham Hospital at 3:30 P.M. there will be a clinic conducted by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, to which physicians are cordially invited.

THE SCIENTIFIC EXHIBIT AT THE CLEVELAND SESSION OF THE AMERICAN MEDICAL ASSOCIATION

Application blanks are now available for space in the Scientific Exhibit at the Cleveland Session of the American Medical Association, June 11 to 15, 1934. The Committee on Scientific Exhibit requires that all applicants fill out the regular application form and requests that this be done as early as convenient.

The final date for filing applications is February 26, 1934. Any persons desiring to receive an application blank, should address a request to the Di-

rector, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago, Illinois.

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DAVID LITTMANN, M.D., announces the removal of his office from 178 Washington Avenue, Chelsea, to 64 Boston Avenue, West Medford, Mass.

A MALARIA LIBRARY

A malaria library was founded in Rome by the Stazione Sperimentale per la Lotta Antimalarica in 1925, and an *Index to Malaria Literature* is issued annually by the Station.

To make this as complete a central malaria library as possible we appeal to all malariologists to send us books, reports and articles on malaria.

Photostat copies of any articles in the library can be had, on request at cost production.

All publications and requests should be addressed to "The Director, Stazione Sperimentale per la Lotta Antimalarica, Corso Vittorio Emanuele, 168, Rome (16)."

A. MISSIROLI, *Director.*

MELANOTIC SARCOMA AND NEUROGENIC FIBROSARCOMA

For research on a diagnostic procedure specimens of blood, tumor tissue and the pituitary gland are desired from cases of these two rare forms of sarcoma. It will be greatly appreciated if physicians having such cases will communicate with Dr. Charles C. Lund, Huntington Memorial Hospital, 695 Huntington Avenue, Boston. Telephone Longwood 4343.

Containers and directions for taking the specimens will be sent.

MASSACHUSETTS GENERAL HOSPITAL

The exercises at the Massachusetts General Hospital on Thursday forenoons are of special interest to physicians. For details see the regular announcement on page viii.

REPORTS AND NOTICES OF MEETINGS

BOSTON PATHOLOGICAL SOCIETY

Regular Monthly Meeting, October 9, 1933. President Tracy B. Mallory in the chair.

Dr. Timothy Leary presented two papers, abstracts of which follow:

1. HUMAN CORONARY AND EXPERIMENTAL RABBIT ATHEROSCLEROSIS: A Comparison of Lesions.

This work was aided by a grant from the Committee on Scientific Research of the American Medical Association.

Cholesterol is a stable, relatively insoluble substance, resistant to enzymes and serving, according to Starling, as the stable substance or framework in the animal cell, in the interstices of which the more labile constituents undergo their metabolism. Egg, milk and pork fats are the human sources of cholesterol, which is practically the only absorbable sterol. The needs for this substance are greatest in times of rapid cell formation. Egg yolk is intended for the embryo, milk for the infant. Man is the only animal that ingests these foods throughout its lifetime, and is the only animal, so far as is known, suffering from fatal coronary sclerosis, and exhibiting high-grade atherosclerosis commonly, especially in young individuals. Moreover, milk and eggs furnish the highest percentage of food sensitization reactions.

Rabbits five months of age were fed cholesterol by catheter over a period of seven months. Four animals were permitted to live five months after cessation of feeding. Spontaneous *medial* sclerosis is not rare in rabbits. Medial sclerosis in human visceral arteries is uncommon. *Intimal atherosclerosis* makes up probably 95% of the sclerotic lesions in these vessels.

Spontaneous *atherosclerosis* (*intimal*) does not occur in rabbits, according to Schoenheimer. Other observers report its possible occurrence in ten out of 3024 rabbits, an incidence of 0.33%. In this experimental series atherosclerosis of the aorta was produced in 25 out of 27 animals, or 92.6%. Rabbits normally live to an age of eight years or beyond. The maximum age of animals at the close of this experiment was seventeen months. Control animals fed equal amounts of sunflower seed oil (which was used as a solvent of cholesterol in the latter half of the period of experimentation), but without cholesterol, showed no evidence of atherosclerosis. This is in accordance with Schoenheimer's observation that plant sterols are not absorbable. An idiosyncrasy with reference to cholesterol was exhibited by these animals, the end results under essentially equal total dosage of cholesterol varying from scattered small aortic lesions in some animals to massive processes including most of the aorta in others. This variation in susceptibility is analogous to that seen in human beings, who may die in the twenties from coronary sclerosis, or live to old age with little evidence of arteriosclerosis.

Most previous studies of experimental atherosclerosis have dealt largely with aortic lesions, and have confused the issue because of the complex character of the aorta histologically. The coronary arteries, per contra, are of relatively simple structure. Moreover, coronary atherosclerosis as a cause of disease and death is of greater importance than aortic sclerosis. Experimental studies permit of observation of the origin and evolution of lesions. Illustrations were shown of imbibition cholesterosis and the advent of large cells—macrophages—which engulf the cholesterol, and come to

rest beneath the endothelium of arteries. Comparative pictures were shown of the massing of these lipoid cells in human and rabbit arteries, the splitting of the cholesterol esters with the production of fibrous tissue, and the formation of atheromatous "abscesses". Lesions were produced in rabbit coronary arteries which could not be differentiated from the natural lesions found in human coronary sclerosis, and included all stages in the process save calcification.

In young rabbits the characteristic form of atherosclerosis is marked by the progressive replacement of the lipoid cells by fibrous tissue. This is also true in young human beings. This standard reaction in youth to the presence of cholesterol in quantity is probably the result of an efficient repair mechanism in the young. In older human beings there is a tendency to the greater massing of lipoid cells with a minimal amount of supporting connective tissue. Because of the poor support and the resulting poor circulation, massive necrosis of the lipoid cells occurs, with splitting of the cholesterol esters and liquefaction. This gives rise to the formation of the so-called atheromatous "abscess," a cavity filled with necrotic detritus, lipoid cells, cholesterol crystals and fluid. It is suggested that fatty acids set free by the splitting of cholesterol esters may stimulate the formation of connective tissue when set free in small amounts progressively, but may cause necrosis when suddenly set free in large quantities.

The evidence presented indicates that the lesions of atherosclerosis as it occurs in the human coronary may be reproduced experimentally by feeding cholesterol to rabbits.

2. Lesions of Fatal Coronary Sclerosis.

The medical examiner is given exclusive opportunity to study the lesions in human beings who die suddenly without apparent adequate cause. This group includes cases of individuals who literally drop dead. The findings in the coronary arteries in these cases are those which immediately produced the death, unmodified by inflammatory and repair pictures such as are found in cases which have been hospitalized before death. Microscopical examination of a large series of coronary arteries indicates that death is usually due to:

1. Fibrosis with narrowing of the coronary lumen. Terminal necrosis of subendothelial connective tissue with extension to the endothelial layer and resulting *thrombosis*. This type of lesion is met with almost exclusively in young persons.

2. Rupture of an atheromatous "abscess" into the lumen through the thinned living "abscess" wall, or through a necrotic region in the wall, with resulting obstruction and *thrombosis*. Reverse rupture through the wall into the "abscess" with thrombosis is possible. This type of lesion is met with almost exclusively in older persons.

3. Narrowing of the lumen, usually due to fibrosis, to the point where the circulation, while adequate under normal conditions, is not adequate under stress. Coronary insufficiency. *No thrombosis.*

This group of causes due to atherosclerosis in the course of the coronary arteries does not include causes of death due to the following: a. aortic insufficiency, with inadequate delivery of blood to the heart during diastole; b. syphilitic stenosis or obliteration of coronary orifices; c. obstruction of orifice by vegetation; d. coronary spasm; e. coronary embolism.

Colored (Agfa) photomicrographs were shown illustrating the occurrence of the pathological conditions met with in atherosclerosis in the course of the coronary arteries, and particularly the factors giving rise to the actual thrombosis.

Gross demonstrations were made of mild and advanced atherosclerotic lesions in the aortae of young experimental rabbits in contrast with the aorta of a normal rabbit of certified age, killed at seven years, 1 month, and showing no atherosclerosis. Gross and microscopical specimens from an athlete, 25 years of age, who died suddenly, in apparent full health, while playing ball, were also demonstrated. The lesions were sclerosis and thrombosis of the descending branch, and fibrous narrowing, almost to occlusion, of the circumflex branch of the left coronary artery, moderately advanced atherosclerosis of the right coronary artery, and focal atrophy of the myocardium with repair (so-called chronic myocarditis), near the apex. The aorta was thin, smooth and elastic, but exhibited one scarred and atheromatous focus in the abdominal vessel.

DISCUSSION

Dr. Paul D. White referred to the historical letter of "that excellent pathologist" Dr. Edward Jenner to Dr. Caleb H. Parry, in which disease of the coronary arteries was first described in England. Jenner, failing to find in the examination of "the more important parts of the heart" anything to account for the sudden death or the symptoms in a case of Heberden's newly named "angina pectoris," stated that in making a transverse section of the heart his knife struck against something so hard and gritty as to notch it. He looked at the ceiling which was old and crumbling, "conceiving that some plaister had fallen down." On further examination, however, he found that "the coronaries were become bony canals". On the basis of this finding he offered to wager that the coronary arteries would be "ossified" in a second case, which Mr. Paytherus had evidently referred for postmortem examination. At the examination, however, though the coats of the artery were hard, "a sort of cartilaginous canal was formed within the cavity of each artery". Dr. White asked if calcareous changes were found exclusively in older persons, or sometimes in younger persons. Had medial sclerosis been produced in this experimental series, and how frequently did it occur in

human coronary arteries? How important is coronary spasm? Did the collateral circulation, when the coronary circulation is interfered with, come from the Thebesian veins or from pericardial vessels? Dr. White discussed the possible relation of alcohol to coronary disease and wondered how it could have an influence.

DR. LEARY: Calcification in any considerable degree in the coronary arteries is unusual under 45-50 years, unlike the findings in the aorta. It always represents a monumental deposit marking the site of what had been formerly living tissue. In other words, it is an end result betokening the termination of progressive lesions. It is common in the old. Medial sclerosis did not arise in this experimental series. It is of minor importance in sclerosis of the coronary arteries in the human being. A few cases have been reported. Coronary spasm is probably responsible for the subendothelial injury and necrosis which leads to endothelial necrosis and thrombosis in the young. A mechanical pressure factor, probably spasm, is responsible for the rupture of atheromatous "abscesses" which perforate through the thinned but living wall of the "abscess" cavity. The media is usually well preserved in coronary atherosclerosis, and may be hypertrophied. Dr. Joseph T. Wearn, with whom Dr. Leary reported two cases of syphilitic obliteration of both coronary orifices, believed at that time that the Thebesian vessels cared for the collateral circulation. Later he discussed anastomoses between aortic and coronary vasa vasora and vessels in the subepicardial fat pads about the aortic ring. His present position Dr. Leary did not know. Discussion of the relation of alcohol to human arteriosclerosis arose from the observation that alcoholics dying at advanced ages showed less sclerosis than was usual at their ages. With reference to coronary disease Dr. George Blumer (personal communication) has stated that, influenced by the large number of total abstainers who suffered coronary disease, he had looked into the history of 150 cases and found only four who were excessive users of alcohol. Cholesterol is relatively insoluble. Among edible substances sunflower seed oil (one of the best solvents), when warm, dissolves one part of cholesterol in 20 of oil. Cholesterol is dissolved by alcohol in a ratio of one to 40. Whether alcohol acts as a solvent, or by its narcotic or depressive action is a question.

Dr. William D. Reid asked what caused the breaking up of cholesterol in the lesions. He referred to a paper by Dr. Raab who advised the avoidance of cholesterol foods as a prophylactic procedure. Wells discussed the occurrence of calcium in the body during life in a semi-solid form, not rigid. Was it possible that calcium existed in this form and became rigid after death?

Dr. Leary stated that cholesterol did not break up so far as is known in atherosclerosis. The cholesterol esters are split setting free cholesterol crystals. Why, is not known. There is a distinct suggestion that a quantitative factor plays a part—minor cholesterosis is common in the aorta in the young (atheromatosis); more marked cholesterosis with lipoid cell infiltration tends to be followed by pathological changes. With reference to calcium, the precipitation of this substance occurs in colloidal granules, believed to be calcium soaps, from which calcium salts become free and are fused in the calcified masses found. Adami referred to vessels with lime salts which were plastic during life but rigid after death. However, beaded or rigid radial arteries are found frequently during life in the aged. Calcification is not an important factor in the stages of development of coronary disease which we should study, i.e., the early stages.

Dr. Samuel A. Levine discussed the problem of the location of coronary thrombosis most frequently in the left coronary or its descending branch within one inch of the orifice. He cited the record of three brothers who suffered from coronary disease, two of whom died from the disease at about the same age and under 50, all with left coronary thrombosis. He wondered whether inheritance or some familial characteristic could account for the idiosyncrasy. He wondered if spasm would be likely to occur in persons who were asleep when the fatal thrombosis arose.

Dr. Leary stated that most of the fatal coronary thromboses occurred in the left coronary artery, so that frequency of occurrence in the same vessel in members of the same family was not significant. It is true that coronary disease is often familial. It is therefore suggested that studies of pressure in the proximal portion of the left coronary should be made. The main branch, the circumflex, comes off almost at right angles near the point of election for thrombus formation and this might influence results. It is possible that some anatomical peculiarity, or an inherited weak cholesterol metabolism, like the inherited tendency to an inefficient carbohydrate metabolism in diabetics, or familial habits in diet might one or all influence the occurrence of coronary disease. With reference to coronary deaths during sleep the possibility of nightmares giving rise to stress conditions should not be overlooked. Alcoholics and epileptics are often asphyxiated by burying their heads in pillows and gradually shutting off access of air. Dr. Leary believes that the normal individual does not asphyxiate under like conditions because he is awakened, perhaps after a nightmare, and changes his position.

Dr. Shields Warren asked about the source of most of the cholesterol in experimental lesions. Did it arise from imbibition, or was it transported by macrophages? When atheromatous "abscesses" rupture in the aorta there is usually no thrombus formation. It has always been thought that cholesterol interfered with fibrin formation. Is the

speed of the circulation in the aorta responsible for the rarity of thrombosis in the aorta?

Dr. Leary stated that he believed the mass of the cholesterol was carried into the subintimal tissues by monocytes. These cells were moderately abundant in the blood and the spleen sinuses. With reference to thrombosis in the aorta, the rupture of atheromatous "abscesses" occurred through small openings usually and the contents were swept away. When a considerable surface is bared, thrombosis follows in the aorta. In the coronary, narrowing of the lumen precedes the rupture, the rupture is relatively large—a tear—the contents are not carried away, and thrombosis follows.

Dr. Edward MacMahon stated that studies of the pressure factors in the proximal portion of the left coronary had been made in Germany and the currents studied. The results had not been conclusive.

Dr. Monroe J. Schlesinger suggested that in alcoholism associated with fatty changes in the liver, the Kupffer cells might be inhibited from engulfing and transporting foreign material, including cholesterol, and that thus atherosclerosis might not result.

Dr. Tracy Mallory referred to Aschoff's theory of the pathogenesis of arteriosclerosis. Deposits of cholesterol in the aorta occur in infancy and early life, are absent in the young adult period, and are again found as the individual ages. The belief that the early process is an almost physiological deposit in a natural reservoir (atheromatosis) and that this cholesterol may disappear when the supply is cut down and the body needs the reserve, is supported by the reports of the absence of cholesterol deposits in the aortae of young persons dying during the post-war famine period in Germany. Associated with an increase of fibrous tissue in the subendothelial tissue of the intima in later life, the process is no longer reversible, and pathological changes characteristic of atherosclerosis develop.

Dr. Leary stated that Aschoff speaks of molecular disintegration of the tissues associated with cholesterol deposits which were becoming pathological, and confessed that he did not know what that phrase meant.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, December 12, at 8:15 o'clock.

PROGRAM

Presentation of Cases.

Chemistry of the Thyroid Hormone. By Dr. W. T. Salter.

Effect of Anterior Lobe Hormones on Thyroid and Parathyroid. By Dr. S. Hertz.

Total and Thyroxin Iodine in Relation to Dosage of Thyroid. By Dr. J. Lerman.

Some Atypical Clinical Pictures of Thyrotoxicosis. By Dr. J. H. Means.

JOHN HOMANS, M.D., *Secretary.*

HOUSE OFFICERS' ASSOCIATION OF THE BOSTON CITY HOSPITAL

MONDAY, DECEMBER 4, 1933

SUBJECT: ROENTGENOLOGY

8:00 P.M. Presentation of cases.
8:15 P.M. Bones—Neoplasms and Infections. Dr. Albert M. Moloney.
8:35 P.M. Treatment—Uses and Limitations. Dr. Frederick W. O'Brien.
8:55 P.M. Chest—Heart and Lung Conditions. Dr. Max Ritvo.
9:15 P.M. Gastro-Intestinal and Renal Tracts. Dr. Paul F. Butler.
9:45 P.M. Discussion.

NOTE: This meeting will be held in the amphitheater of the new pathology building which is opposite the main hospital at the corner of Albany Street and Massachusetts Avenue.

ROBERT T. PHILLIPS, M.D., *Secretary.*

NEW ENGLAND PEDIATRIC SOCIETY

The next meeting of the New England Pediatric Society will be held on Friday, December 15, 1933.

AFTERNOON SESSION—BOSTON CITY HOSPITAL

4:00 to 6:00 P.M. Clinic. Dr. Martin English.

EVENING SESSION—BOSTON MEDICAL LIBRARY

8:15 P.M. Election of Officers.
Awarding of New England Pediatric Society Prize.
Speaker: Dr. Edwin H. Place—"Septic Sore Throat."

GERALD HOEFFEL, M.D., *Secretary.*

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society, of the Tufts College Medical School, will be held at 8:00 P.M. on December 8, in the auditorium of the Beth Israel Hospital, Boston.

The speaker will be Dr. Harrison S. Martland, Pathologist to the Newark City Hospital. Subject: "The Causes of Sudden Death." Chairman: Dr. Timothy Leary, Medical Examiner, Suffolk County, Southern District.

Members of the medical profession are cordially invited to attend the meeting.

FAULKNER HOSPITAL CLINICAL MEETING

The next meeting will be held at The Faulkner Hospital at 5:00 P.M. on Thursday, December 7. In addition to the usual pathological conference on the cases which have come to autopsy during the month Drs. F. J. Cotton and Henry C. Marble will talk on "Traumatic Surgery."

BOSTON CITY HOSPITAL

STAFF CLINICAL MEETING

Wednesday, December 6, 1933, at 8:15 P.M.
Thorndike Amphitheatre

Note Change of Date

Nephrectomy in Renal Tuberculosis and the Value of Long-Continued After-Care. Dr. Herbert H. Howard. (Discussion by Dr. George Gilbert Smith.)

Nephropexy for Ureteral Kinks. A Review and Results of Ninety Cases. Dr. Augustus Riley. (Discussion by Dr. J. Dellinger Barney.)

Perineal Injection of Mercurochrome in Prostatic and Vesicular Infections. Dr. Gordon D. Atkinson.

Treatment of Acute Gonorrheal Arthritis of Knee. Dr. William F. Cotting. (Discussion by Drs. H. H. Howard, A. Riley and O. J. Hermann.)

COMMITTEE ON HOSPITAL CLINICS.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

December 4—House Officers' Association of the Boston City Hospital. See page 1135.

December 6—Boston City Hospital, Staff Clinical Meeting. See notice above.

December 7—Clinic at the Peter Bent Brigham Hospital. See page 1131.

December 7—Faulkner Hospital Clinical Meeting. See page 1135.

December 8—William Harvey Society. See page 1135.

December 12—Harvard Medical Society. See page 1135.

December 13—New England Dermatological Society will hold its next meeting at 3 P.M. at the Boston City Hospital.

December 15—New England Pediatric Society. See page 1135.

December 15 and 16—The American Board of Dermatology and Syphilology will hold oral examinations in New York City. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

December 17, January 15—Boston Medical History Club.
Program:
December 17, 1933—Professor Norman E. Himes, of Colgate University, "Medical History of Contraception."
January 15, 1934—Professor Henry E. Sigerist, Director of the Institute of Medical History of Johns Hopkins University.

JAMES F. BALLARD, Secretary.
Boston Medical Library, 8 The Fenway, Boston.

February 16 and 17—The New England Hospital Association is holding its Twelfth Annual Meeting at the University Club, Boston. For details write Dr. A. G. Engelbach, Massachusetts General Hospital, Boston.
April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.
July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.
September 4, 5, 6, 1934—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Wednesday, December 6—Beverly Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Henry Pollock, Superintendent of Massachusetts Memorial Hospitals. Subject: "Some Problems in the Administration of the General Hospital."
Wednesday, January 3—Danvers State Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Clarence A. Bonner, Superintendent of Danvers State Hospital. Subject: Recent Experiences in Foreign Clinics.
February 7—Council Meeting, Boston.
Wednesday, March 7—Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Frank H. Lahey, Boston. Subject to be announced. Film Electrocardiogram.
Wednesday, April 4—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speakers: Dr. Elliott P. Joslin and Dr. Howard F. Root, Boston. Subject: "Tuberculosis Complicating Diabetes."
Thursday, May 3—Censors' Meeting, at Salem Hospital, 3:30 P.M.
Tuesday, May 8—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker to be announced. Subject to be announced.

RALPH E. STONE, M.D., Secretary.
221 Cabot Street, Beverly, Mass.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland, Mass.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings will take place on January 10 at Melrose; March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.
76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on January 31, and April 25 (Annual Meeting).

T. A. STAMAS, M.D., Secretary.
226 Central Street, Lowell, Mass.

NORFOLK DISTRICT MEDICAL SOCIETY

January 28—Tentatively Roxbury Masonic Temple, 8 P.M. Continuance of Symposium on Diseases of the Blood. Dr. Dameshek and Dr. Straus.
February 27—Place, time, subject, and speaker to be announced.
March 27—Place and time to be announced. Dr. Edward A. Edwards and Dr. Henry H. Faxon. Symposium on the Treatment of Varicose Veins.
May—Annual Meeting. Place, time and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline, Mass.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

December 7—12 noon at Quincy City Hospital. Program by the hospital staff.
January 4—12 noon at Norfolk County Hospital. Speaker to be announced.
February 1—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.
March 1—12 noon at Quincy City Hospital. Program by the hospital staff.
April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.
May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.
Norfolk County Hospital, South Braintree, Mass.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 31—General Meeting in association with the Boston Medical Library and the Trudeau Society. Speaker: Dr. Lawrason Brown of Saranac Lake.
March 28—Clinical Meeting at the Massachusetts Memorial Hospitals.
April 25—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program, titles and speakers to be announced.
The Medical Profession is cordially invited to attend all of these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary.
311 Beacon Street, Boston, Mass.
ALEXANDER S. BEGG, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:
December 13—Dinner and scientific program at St. Vincent Hospital, Worcester, Mass.
January 10—Dinner and scientific program at the Worcester City Hospital, Worcester, Mass.
February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.
March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.
April 11—Open date.
May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester, Mass.